A PROSPECTIVE OPEN LABELLED NON RANDOMISED PHASE-II CLINICAL TRIAL ON

THANDAGA VATHAM

(LUMBAR SPONDYLOSIS)

WITH

MUNNAI ILAI KUDINEER

Dissertation submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY, CHENNAI–32

For the partial fulfillment of the requirement for the degree of

DOCTOR OF MEDICINE (SIDDHA)

(Branch-I Pothu Maruthuvam)

DEPARTMENT OF POTHU MARUTHUVAM

GOVERNMENT SIDDHA MEDICAL COLLEGE

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OCTOBER 2019

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This is to certify that the dissertation entitled **"A PROSPECTIVE OPEN LABELLED NON RANDOMISED PHASE-II CLINICAL TRIAL ON THANDAGAVATHAM (LUMBAR SPONDYLOSIS) WITH MUNNAI ILAI KUDINEER"** is abonafide work done by **Dr. N.PRAKASH (Reg. No.321611007)** Govt. Siddha Medical College, Palayamkottai in partial fulfilment of the University rules and regulations for award for **MD (S), BRANCH-I POTHU MARUTHUVAM** under my guidance and supervision during the academic **year OCTOBER 2016-2019.**

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Certified that I have gone through the dissertation entitled "**A PROSPECTIVE OPEN LABELLED NON RANDOMISED PHASE-II CLINICAL TRIAL ON THANDAGA VATHAM (LUMBAR SPONDYLOSIS) WITH MUNNAI ILAI KUDINEER"** submitted by **Dr. N.PRAKASH (Reg. No.321611007) a student of final year MD (S), Branch-I, Department of Pothu Maruthuvam** of this college and the dissertationwork has been carried out by the individual only. This dissertation does notrepresent or reproduce the dissertation submitted and approved earlier.

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ACKNOWLEDGEMENT

First of all I thank my Almighty God for his showered blessings upon me in performing my dissertation works. I express my gratitude to **Vice-Chancellor, the Tamil Nadu Dr. M.G.R. Medical University,** Chennai and **Special Commissioner, Commissionerate of Indian Medicine and Homeopathy,** Chennai for granting me to undertake this dissertation work.

My sincerely thank to **Prof. Dr.S.Victoria,M.D(S),** Principal, Govt. Siddha Medical College, Palayamkottai for permitting me to use all the facilities available in this institution.

I extend my sincere thanks to Former Principal **Prof. Dr. R.Neelavathi, MD(S),Ph.D,**Govt. Siddha Medical College, Palayamkottai for their approval and support provided as the for runner of the study.

I also wish to express my sincere gratitude to my supervisor **Prof.Dr.A.Manoharan,MD,(Ph.D)** Head Department of Pothu Maruthuvam, Government Siddha Medical College, Palayamkottai, Tirunelveli for his encouragement, patience, guidance and his excellent supervision during my stay at the Department.

I express my deep gratitude to **Dr. T.Komalavalli, MD (S), Ph.D.,** Associate Professor, Guide, Post Graduate Department of Pothu Maruthuvam for her devoted guidance in my dissertation work.

I would take this moment to signify my sincere gratitude to **Dr. S.Justus Antony, MD(S), Dr. G.Subash Chandran, MD (S), Ph.D., Dr. S.Uma Kalyani, MD (S), Dr. P.Sathish Kumar, MD (S),** Lecturers Post Graduate Department of Pothu Maruthuvam for their valuable suggestions for my dissertation work.

I express my thanks to **Dr. (Mrs). S.Sutha, M.Sc., Ph.D.,** Associate Professor in Department of Medicinal Botany, Govt. Siddha Medical College, Palayamkottai for her suggestions in the botanical aspect of my work.

I thank **Prof. Mrs. N.Naga prema, M.Sc., M.Phil.,** and other technical staffs, Department of Biochemistry for helping me in elicting biochemical analysis.

I express my thanks to **Mrs. T.Poonkodi, M.A., M.L.I.S.,** Librarian, Govt. Siddha Medical College, Palayamkottai for permitting me to utilize the college library.

I express my thanks to all lab technicians in helping me to elicit the haematological investigations.

I express my thanks **Dr.N.Chidambaranathan, M.Pharm, Ph.D,** Vice principal, K.M.College of Pharmacy, Madura**i** who helped me in carrying out the pharmacological Study of the clinical trial medicine.

Also, I express my gratitude to Inbiotics (Affliated to Institute of biology and clinical research), Nagarcoil for the Antimicrobial Study of the trial medicine.

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LIST OF ABBREVIATIONS

%	-	Percentage
ALT	-	Alkaline Phosphatase
ANOVA	-	Analysis of Variance
AS	-	Ankylosing Spondylitis
ASO	-	Anti Streptolysin 'O' Factor
B_1gene	-	Beta 1 gene
Bid	-	Twice aday
BMI	-	Body Mass Index
BPFS	-	Back Pain Functional Scale of Stratford et al
CT	-	Computerized Tomography
CM	-	Centimeter
CRP	-	C-Reactive Protein
DC	-	Differential Count
DL	-	Decilitre
E	-	Eosinophilis
EMG	-	Electromyography
ESR	-	Erythrocyte Sedimentation Rate
GMS	-	Grams
HB	-	Haemoglobin
HDL	-	High Density Lipoprotein
i.e.,	-	Thatis
IGC	-	Intra Gastric Catheter
IVD	-	Inter Vertebral Disc
JVP	-	Jugular Venous Pulsation
KG	-	Kilo Grams
L	-	Lymphocytes
L_5-S_1	-	5^{th} Lumbar Vertebrae upto first sacral vertebrae
MG	-	Milli grams
ML	-	Milli Litre
MM	-	Milli Meter
MRI	-	Magnetic Resonance and Imaging
MS	-	Multiple Sclerosis

NPRS	-	Numeric Pain Rating Scale
^{o}	-	Degree
ODI	-	Oswestry Disability Index
OPLL	-	Ossified Posterior Longitudinal Ligament
P	-	Polymorphs
PSEQ	-	Pain Self-Efficacy Questionnaire
PSFS	-	The Patient-Specific Functional Scale
RA	-	Rheumatoid Arthritis
RBC	-	Red Blood Corpuscles
REF	-	Reference
RMDQ	-	Roland Morris Disability Questionnaire
SAP	-	Superior Articular Process
SEM	-	Structural Equation Modelling
SLE	-	Systemic Lupus Erythematosus
SLR	-	Straight Leg Raising
SPECT	-	Single Photon Emission Computed Tomography
T_9-T_{10}	-	9^{th} Thoracic Vertebrae to 10^{th} Thoracic Vertebrae
TC	-	Total Count
U/L	-	Units per Litre
WBC	-	White Blood Corpuscles
WHO	-	World Health Organisation

ABSTRACT

Low back pain (LBP) is one of the major hazards to mankind. Now a days people sit early morning at lavatory and end up their day sitting in the dining table for dinner. They have not time to walk or take a nap. Some people toil all through the day standing and bending. They have no time to sit or sleep. This ultimately results in low back pain which affects approximately 60%-85% of adults. Low back pain which is the chief complaint of Thandaga Vatham, mentioned in Siddha text book Yugi Vaidhya Cinthamani-800, which is correlated with the clinical features of Lumbar spondylosis has become a major problem today, despite of many advances in treatment. Conventional medicines and surgical treatments end up with side effects and leave with defect and detriment like recurrence of pain, disability & nerve root damage.

This study is concerned with 'A Prospective open labelled non randomised Phase-II Clinical trial to assess the therapeutic efficacy of the siddha formulation **MUNNAI ILAI KUDINEER** for the treatment of **THANDAGA VATHAM** (LUMBAR SPONDYLOSIS)', described in Gunapadam Mooligai Vaguppu Part-I text book to detect whether any significant improvements and relief can be done. Totally 40 patients with Thandaga Vatham (20 In patients, 20 Out patients of both sex) were selected randomly from OPD of Pothu Maruthuvam, Govt. Siddha Medical College, Tirunelveli, Tamil Nadu. They were given Munnai Ilai Kudineer 30ml twice a day for 30 days. After the course of treatment majority of cases showed good response which is statistically significant.

Keywords:
Thandaga Vatham, Lumbar Spondolysis, Munnai Ilai Kudineer, Cardinal Signs, Range of motion, Back Pain Functional Scale Score (BPFSS).

CHAPTER-I

INTRODUCTION

The siddha system is one of the ancient systems of medicine.This system hasbeen cured various disease, including Mukkutra noigal, Peru noigal, Gunmam, Auto immune disorders and skin diseases.The fundamental and classical features of siddha treatment is to destroy the root cause of the diseases and it will strengthen the body, mind and soul.Siddha system of medicine is not only cure diseases but also has preventive and rejuvenation of the health .Siddha system of medicine differs from other systems, because the etiology management and of diseases. The main objective of the treatment is to equilibrium of three humours, namely Vatham, Pithamand Kapam**.**The cretantials of treatment is based upon balancing the three humours.The disturbances of the vatham which may be reduced or exaggerated lead to vatha diseases.

The YUGI VAITHIYA CHINTHAMANI-800 is classified into 4448 types. Among 4448 diseases the Vatha diseases are 80 types and Pitha diseases are 42 and kapamdiseases are 21 types. Thefive elements (Pnachabootha panchikarnams) possess two properties viz., subtle and gross. These elements always act in mutual co-ordination and can never act independently. The various proportions in which they combine give rise to different substances. Thus, this theory proposes that 96 basic factors exist, which is the basic concept underlying this holistic medical science.

The human body formed by these 96 basic factors is conditioned mainly by Uyir thathukkal and Udal thathukkal.The 96 factors are the physical, physiological, psychological, intellectual aspects of every human. The five primordial elements manifest themselves as a human with these 96 basicfactors.

Raw drugs are classified based on maintaining the three vital humours as Thavaram (Plants), Thathu (Metals and Minerals) and Jeevam Products (Animal Products).Further, drugs are based on Suvai (Taste), Gunam (Character), Veeriyam (Potency) and Pirivu (Section). The thandaga vatham is one among the vatha disease,it was coming under the head of degenerative disease .The clinical symptoms and signs is similarly corrlated in modern medicine is , Lumbar spondylosis.

In India, Lumbar spondylosis affects 60%-85% of the adults during some point in their lives. 84% of men and 74% of women are suffering from Lumbar spondylosis in the age group of 45-64 years of population is prone to develop

disorders of vertebral column ,like lumbar spondylosis Prolapsed Intervertebral DiscPID),osteoporosis and the degenerative disease of spine.Majarity of them are suffering from PID or lumbar spondylosis. The clinical features of Lumbar spondylosis is Pain from the low back, sacroiliac region and ascend lower extremitiess The compersson of roots,can produced superficial and deep sensory symptoms. There is no proper medication available in other systems.But siddha system was mentioned in various text books and manuscript to treat the thantagavatham. This work is to deals elaborately discussed about the etiology, pathology, clinical feature, external and internal treatment and complications of Thandaga Vatham.So, the clinical trial drug **"MUNNAI ILAI KUDINEER**" is mentioned in **GUNAPADAM MOOLIGAI VAGUPPU PART-I** is more effective to reduced back pain and strengthen the para spinal muscles.In review articles are proved Munnaii Ilai has good anti-inflammatory, analgesic and anti-vatha activities and pharmacological research also proved the same.

CHAPTER-II

AIM AND OBJECTIVES

AIM:

To evaluate the clinical and therapeutic efficacy of **Munnai Ilai Kudineer** in **Thandaga Vatham (Lumbar Spondylosis).**

OBJECTIVES:

Primary Objective:

- To develop evidence support for the effect of Munnai Ilai Kudineer in ThandagaVatham.

Secondary Objective:

- To evaluate the Anti inflammatory and Analgesic Pharmacological activity of Munnai Ilai Kudineer.
- To determine the safety profile of clinical trialdrugs.
- To evaluate the Bio chemical analysis in Munnai ilia kudineer.
- To survey the incidence of the disease, according to Age, Occupation, Socio Economic Status, Habits, Family History, Paruva Kaalangal, Thinai and Three Vital Humours.
- To evaluate the changes in the Envagai thervugal in Thandaga Vatham according to Siddha basic concepts.
- To carried out Modern parameter changes in Thandaga vatham (Lumbar Spondylosis).
- To discuss about prognosis and treatment effect after end of the study.

CHAPTER-III

REVIEW OF LITERATURE

3.1 IN JOURNAL ASPECT:

Anti inflammatory Activity

Karthikeyan and Deepa. 2011 studied reports showed that the PCEE significantly inhibited the egg albumin induced edema and reduced granuloma formation in cotton pellet method and have preferable anti-inflammatory effect with long term administration. The phytochemical studies revealed the presence of β-sitosterol and luteolin in leaves. The anti-inflammatory activity may be due to the presence of β-sitosterol or luteolin. Luteolin exerted the strongest blocking action on expression of this inflammatory mediator Luteolin, exerted inhibitory effects on TNF-alpha, IL-6 and IFN-gamma production Luteolin suppressed TNFalpha-induced IL-8 production in dose-dependent manner.

Antihyperglycaemic activity

Dash et al. 2005 was studied on antihyperglycaemic activity of Premna corymbosa reveal that the extract of the leaves showed significant antihyperglycaemic activity against alloxan induced diabetic rats, when compared with the controls. However, in normoglycaemic animals, there was not much marked decrease observed in the blood sugar level at tested dose levels.

Hepatoprotective activity

Karthikeyan and Deepa were studied the histopathology of the liver sections evidenced the hepatoprotective activity. The liver weight was reduced by the ethanolic extract treated groups.The ethanolic extract of the leaves of *Premna corymbosa* possess significant acute hepatoprotective activity. *Premna corymbosa* can be recommended for the liver disorders.

Cardiac Stimulant Activity:

Rajendran et al. 2008 Cardiac stimulant activity was reported for water and ethanol extract of bark and wood of ***Premna corymbosa***. The ethanol extract produced significant positive ionotropic effects similar to that of Digoxin. A significant decrease in membrane Na+K +ATPase and Mg2+ ATP ase and significant positive ionotropic and chronotropic effects similar to that of Adrenaline. It was concluded that the ethanol extract produced cardiotonic effect and the aqueous extract produced adrenergic effect.

Antitumor Activity:

Serrame and Limsylianco.1995 Anti-tumor promoting activity of decoctions and expressed juices from some philippinel medicinal plants including ***Premna corymbosa*** was evaluated. Significant inhibition of growth of tumours was shown by ***Premna corymbosa*** .

Antibacterial Activity:

Anbazhakan and Bahu, 2007 Studied showed Antibacterial activity of hexane, chloroform, ethyl alcohol and water extracts and water extracts of the stem bark of ***Premna corymbosa*** was done and it was reported that chloroform extract of the root inhibited the growth of Entero bacteraero genes but there was no activity on the other extracts. The presence of Di-C-glycosyl flavones in the heart wood of plant might be the reason for antibacterial activity. Also all the extracts were active against Alkaligens faecalis, Bacillus subtilis and Eischeria coli .

Diuretic Activity:

Anbazhakan and Babu, 2007 was studied Alcoholic extract of roots of Premna latifolia was found to show diuretic activity. The urine output was more in rats treated with 1mg/kg and 2mg/kg body weight and also in dogs treated with 2mg/kg than the control group. The mechanism of action was thought to be direct vasodilator effect (Rema and Vijayamma, 1995).A study on indigenous knowledge of Premna tomentosa was done. It of was concluded that leaves this plant has diuretic properties and can be used in dropsy treatment. Also an extract of inner bark was used to arrest diarrhea and the decoction of root can be given in stomachache. It was also used by people for curing rheumatism, liver and spleen disorders and joint pains .

Antiparasitic Activity:

Desrivit et al. 2007 was done antiparasitic activity of some new caldonian medicinal plants including *Premna corymbosa* were evaluated. It was observed that Premna serratifolia was active against Leishmania donovani with IC50 values between 0.5- 5µg/ml .

3.2 IN SIDDHA LITERATURE

3.2.1 IN GUNAPADAM ASPECT- MUNNAI

Tamil name : Munnai

Other names : Arani, Vaisayanthi

Suvai : Thuvarpu, Kaippu

Thanmai : Veppam

Privu : Karppu

Part used : Ilai, Ver

Actions

1. Stomachic
2. Carminative
3. Alternative
4. Tonic

FIGURE-1

FIGURE-2

Tamil Name	Botanical Name (Family)	Phytochemicals	Action	Therapeutic uses	Amount
Leaves of **Munnai**	*Prema corymbosa* (VERBENACEAE)	Verbascoside, Iridoid glycoside Premcoryoside Premnine ,Premnazole Premnenol Premna spirodiene .	Anti-inflammatory, Anti-oxidant, Anti-analgesic	Vatha disease	7.5gms

3.2.2 SIDDHA ASPECTS:

The concept of Siddha system of medicine is based on 96 Thathuvangal which is made up of five primordial basic elements.Among which the three vital humours, Arusuvaigal and udal thathukkal play an important role.The vatham is mainly affected in in thandagavatham.Vatham is characteristic by vayu, dryness, pain, flatulence, sensitiveness, lightness, and also air.

Vatham has a vital role in locomotion (or) movement activity. If Vatham is disturbed, locomotion is affected. The other two humours are deranged on severity of disease. Thus in Noi Naadal and Noi Mudhal Naadal Part-I text book clearly explains Vatham is soverign. It is called as Pranam, of living beings.

வாதமாய் படைத்து
பித்த வன்னியாய் காத்து
சிலேத்தும சீதமாய் துடைத்து

- நோய் நாடல் நோய் முதல் நாடல் திரட்டு
(பாகம்-1, பக்கம் எண்.97)

The Vatha dosham quoted in various Siddha literature are as follows:

According to Agasthiyar,

காணப்பா வாதமீறில் கால்கைகள் பொருத்து நோவும்
பூணப்பா குடல் புரட்டும் மலசலம் பொருமிக்கட்டும்

- அகத்தியர் வைத்திய காவியம் 1500
(பாடல் எண்.10, பக்கம் எண்.2)

According to Thirumoolar,

எறிய நல்வாதம் எறிக்கும் குணங்கேளு
குறியெனக் கைகால் குளைச்சு விலாச் சந்து
புறியென நொந்துடல் பச்சைப்புண் ஆகுமே

- திருமூலர் கருக்கிடை வைத்தியம்-600
(பாடல் எண்.36, பக்கம் எண்.11)

According to Theriyar,

தக்கவாயு கோபித்தால் சந்துவுளைந்து தலைநோவா
மிக்க மூரி கொட்டாவி விட்டங்கெரியு மலங்கட்டும்
ஒக்கநரம்பு தான்முடங்கு முலர்ந்து வாய்நீரூறிவரும்

- தேரையர் வாகடம்
(பாடல் எண்.2, பக்கம் எண்.13)

வாதவீறு அன்னமிறங்காது கடுப்புண்டாம் வண்ணமுண்டாம்
மோதுகட்டுரோகம் சுரமுண்டா மிருமலுமா முறங்காதென்றும்
ஒதுசூரிய வாதமனலாகு நடுக்கமுண்டாம் பொருள்களாய்ந்
தீதெனவே நரம்பிசித்து சந்துகள்தோறும் கடுக்குந் தினமுந்தானே

- தேரையர் வாகடம்

(பாடல் எண்.210, பக்கம் எண்.58)

According to Sivavakiyar, Vatham is Omnipotent. It is the beginning, benevolent, soverign, eminent diety. It has the character to form other twocombained humours.

வாதமான தேவனே யாதியாகி நின்றவன்
வாதமான தேவனே வையகம மைத்தவன்
வாதமான தேவனே யறுதொழில் வகுத்தவன்
வாதமான தேவனே வண்மை கண்டு கூறுமே

- சிவ வாக்கியர் நாடி

The Vatham is primary deranged and further secondary Pitham and Kapam gets affected

வாதமிகு குணம்:

அறியவிம் மூன்றின் தன்மை சொன்னார்நந்தி
ஏறிய நல்வாத மெறிக்குங் குணங்கேளு
குறியெனக் கைகால் குளைச்சு விலாச்சந்து

- திருமூலர் கருக்கிடை வைத்தியம்-600

வாதவீறு அன்னமிறங் காது கடுப்புண்டாம் வண்ணமுண்டாம்
மோதுகட்கு ரோகம் சுரமுண்டா மிருமலுமர முறங்காதென்றும்
ஒதுதரிய வாதமனலாகு நடுக்கமுண்டாம் பொருள் களயர்ந்த
தீதெனவே நரம்பித்து சந்துகள் தோறுங் கடுக்குந் தினமுந்தானே

- தேரையர் வாகடம்

(பாடல் எண்.210, பக்கம் எண்.58)

தக்கவாயு கோபித்தால் சந்துளைத்து சூலைநோவா
மிக்க கொட்டாவி விட்டங் கெரியு மலங்கெட்டும்
ஒக்க நரம்புதான் முடங்கு மலர்ந்து வாய் நீரூறிவரும்
மிக்க குளிரும் நடுக்கமாய் மேனி குன்றி வருங்கானே

- தேரையர் வாகடம்

(பாடல் எண்.43, பக்கம் எண்.13)

வாதம் மிகும் போது பசியின்மை, உடல் கடுப்பு, சுரம், இருமல், உறக்கமின்மை, சுரம் நடுக்கம், நரம்புத் தளர்ச்சி,**சந்துகள்தோறும் குடைதல், விலாச்சந்துகள் நோதல்,** வயிறு பொருமல், குடலிறைச்சல், மலச்சிக்கல், மிகுந்த கொட்டாவி போன்ற குறிகுணங்கள் தோன்றும்..

According to Siddha Maruthuvanga Churukkam, Yugi Munivar says, the Vatham islives from Abanan (Pelvic plexus),Idakalai, Umblical cord, Skin, Joints, Motion, Lower abdomen, Hipbones, Nerves, Hair follicles and Muscles.It was mentioned Siddha maruthuvanga chrukkam in below lines,

நாமென்ற வாதத்துக் கிருப்பிடமே கேளாய்
நாபிக்குக் கீழென்று நவில லாகும்

- சித்த மருத்துவாங்க சுருக்கம்
(பக்கம் எண்.140)

3.3.THANDAGA VATHAM

SYNONYMS:

தண்டுவாதம், இடுப்பு வாதம் (வாத நோய் மருத்துவம், பக்கம் எண்.114, 126)

DEFINITION:

Thandaga Vatham is a clinicalcondition which is characterized by great prostration, in which the body is rendered like a log of wood, unable to stretch or fold the limband passed motion or urine. The whole body assumes rigidity as the stiffness appearing after death. (T.V.சாம்பசிவம் பிள்ளை, அகராதி பாகம்-IV)

AETIOLOGY:

The factors that play its role in modification of Vatham are,

- Environmental factors
- Physical Factors
- Factors of Kanmam

1) Environmental factors (Seasonal Variations):

ஆடியாதியாய் ஐப்பசிஈறாய்
ஆனிலமதற் கோரரசியல் காலம்

- சதகநாடி (நோய் நாடல்
நோய் முதல் நாடல் பாகம்-I)
(பக்கம் எண்.167, 168)

The **Sathaga Naadi** described, the Vatha disease get predominant in the month of Aadi to Iypasi (July to November).

வாத வர்த்தனை காலமேதோ வென்னில்
மருவுகின்ற ஆனி கற்கடகமாகும்
ஆதவைப் பசியோடு கார்த்திகை தன்னில்
அருடமே.....

- யூகி வைத்திய சிந்தாமணி

(பாடல் எண்.245, பக்கம் எண்.76)

According to **Yugi Vaidhya Chinthamani-800** discribed the Vatham is provoked in its own site in the month of Iypasi and Karthigai (வேற்று நிலை வளர்ச்சி) and retains normal in the rest of the month (தன்னிலை அடைதல்).

பதுமத்தைப் பூக்க வைக்கும் பானுமிக்க காயும்
முதுவேனி லிற்பு விநீர் முற்றும் - கதுமென
வற்றும் கபகும் வாயுமிகும் .....

- மருத்துவ தனிப்பாடல்

மேற்கூறிய பாடலின் மூலம் முதுவேனிற் காலத்தில், சூரிய வெப்பத்தின் காரணமாக பெருவாரியாக நீர் ஆவியாக்கப்பட்டு பூமியில் வறட்சி நிலவும். அதுபோல் நமது உடலில் வறட்சி ஏற்பட்டு வளி நோய் வருவதற்கு காரணமாகிறது.

2) Physical factors:

1). According to Sabapathy Manuscript is explained,

வளிதரு காய்கிழங்கு வரைவிலா தயிலல் கோழை
முளிதரு போன்மிகுக்கு முறையிலா வண்டி கோடல்
குளிர்தரு வளியிற் தேகங்குளிப்புற வுலவல் பெண்டிர்
களிதரு மயக்கம் பெற்றோர் கடிசெயல் கருவியாமால

- சபாபதி கையேடு-சித்தமருத்துவம் (பொது)

(பக்கம் எண்.624)

The above poem was explained, the Excessive oral intake of rhizomes and vegetables can are increased Vatha diseases. Irregular food intake, prolonged exposure to cold air, staying in hilly area, excessive sexual activity and hereditary factors also to produced the Vatha diseases.

2). According to Noi Naadal and Noi Mudhal Naadal Part-I, text

Sour and Astringent food stuffs increases the Vatha disease.

மாத்திய புளிப்பு மீறில் வந்திடும் வாதமாகும்
சேத்துமந் தண்ணீர் பித்தந் தீ காற்று வாதமாமே

(அகத்தியர் நாடி)

- நோய் நாடல் நோய் முதல் நாடல்

(பாகம்-I, பக்கம் எண்.22)

Intake of large amount of sour foods increases Vatham.

வாதமே புளிப்பு வேண்டும் வன்பித்தங் கசப்பு வேண்டும்
தீதிலாசி லேற்பனந்தான் சேர்ந்திடும் இனிப்பு வேண்டும்
ஓதிய வாத பித்த சிலேற்பன தொந்தத் தோர்க்குக்
காதலாய்த் துவர்த்தல் காரல் உவர்ப்போடு கருதுங்காணே

(இரத்தினச் சுருக்க நாடி)

- நோய் நாடல் நோய் முதல் நாடல்

(பாகம்-I, பக்கம் எண்.22)

The sour foods would definitely increased the Vatham while astringent foods are added unto it.

3). According to Yugi Muni in Yugi Vaithya Chinthamani-800.

பகரவே வாதமது கோபித்தப்போ
பண்பாக பெண்போகம் அதுதான் செய்யில்
நகரவே வெகுதூர வழி நடக்கில்
நளிரான காற்றுமே பனிமேல் பட்டால்
மிகரவே காய்கள் கனிகிழங்கு தன்னை
மிகவருந்தி மீறியே தயிர்தான் கொண்டால்
முகரவே முதுகெலும்பை முறுக்கி நொந்து
முழங்காலும், கணுக்காலும் கடுப்புண்டாமே

- யூகி வைத்திய சிந்தாமணி-800

(பாடல் எண்.285, பக்கம் எண்.89)

Excessive sexual activity (or) desire, walking for a long distance, prolongs exposure to cold extreme dampness, Intake of harmful food stuffs like excessive curd consumption after eating fruits, vegetables and tubers produce toxic factors which affect muscles and bones produce Vatha diseases.

என்னவே வாதந்தா வெண்பதாகும்
இகத்திலே மனிதர்களுக் கெய்யுமாறு
பின்னவே பெண்தனையே சோரஞ்செய்து
பெரியோர்கள் பிராமணரைத் தூடணித்தும்
வன்ன தேவச் சொத்தில் சோரஞ் செய்து
மாதா பிதா குருவை மறந்த பேர்க்கும்
கன்னவே வேதத்தை நிந்தை செய்தால்
காயத்திற் கலந்துடுமே வாதந்தானே

- யூகி வைத்திய சிந்தாமணி-800

(பாடல் எண்.243, பக்கம் எண்.76)

Breach of trust, abusing the pious, elderly people, priests and holy spirits, exploitation of charitable properties, ingratitude towards mother, father and teacher results in Vatha diseases.

தானென்ற கசப்போடு துவர்ப்பு கைப்பு
சாதகமாய் மிஞ்சுகினும் சமைத்த வண்ணம்
ஆனென்ற ஆறினது புசித்த லாலும்
ஆகாயத் தேறலது குடித்தலாலும்
பானென்ற பகலுறக்க மிராவிழிப்பு
பட்டினியே மிகவுறுதல் பாரமெய்தல்
தேனென்ற மொழியார் மேற்சிந்தை யாதல்
சீக்கிரமாய் வாதமது செனிக்குந்தானே

- யூகி வைத்திய சிந்தாமணி-800

(பாடல் எண்.244, பக்கம் எண்.76)

Excessive intake of bitter, astringent and salty foods, intake of dry and old cooked rice, drinking polluted rain water, irregular sleep patterns, undue starving, excessive weight lifting and sexual perversion can induced Vatha diseases.

ஆனான வறன் றன்னையே மதியா மாந்தர்
அகதி பரதேசியர் கட் கன்ன மீயார்
கோனான குருமொழியை மறந்தபேர்கள்
கொலைகளவு பொய்மங் குறித்த பேர்க்கு
உளனான சடந்தன்னில் வாதம் வந்து
உடற்பவிக்கும் வேதத்தி லுண்மை தானே

- யூகி வைத்திய சிந்தாமணி-800

(பாடல் எண்.253, பக்கம் எண்.95)

Disobedience attitude towards God, refusing food for destitute and Sanyasi,disagreeing the advice of preceptors, engage in murdering, stealing, unjustice and speaking lie leads to Vatha diseases.

4). According to Theraiyar in Theraiyar Vagadam text,

வெய்யிலில் நடக்கையாலும் மிகத் தண்ணீர் குடிக்கையாலும்
சேய்யிழை மகளிரைச் சேர்ந்தன பலிக்கையாலும்
பையனே உண்மையாலும் பாகற்காய் தின்கையாலும்
தையலே வாதரோகம் சனிக்கு மென்றறிந்து கொள்ள

- தேரையர் வாகடம்

(பாடல் எண்.16, பக்கம் எண்.5)

Excessive walking in hot sun, excessive intake of water, excessive sexual activity, intake of bitter guard may disturb the normal functions of Vatham.

5). According to Pararasa Sagaram,

தொழில்பெறு கைப்புக் கார்த்தல் துவர்த்தல் விஞ்சுகினுஞ் சோறும்
பழையதாம் வரகு மற்றைப் பைந்திணை யருந்தி னாலும்
எழில்பெறப் பகலு றங்கி இரவினி லறங்கா தாலும்
மழைநிகர் குழல னாளே வாதங்கோ பிக்குங் காணே

Improper dietary habits and sleep pattern can cause Vatha diseases.

காலங்கண் மாறி யுண்ணுங் காரியத் தாலுந் தண்ணீர்
சாலவே யருந்தி னாலுஞ் சந்தியி லுட்கார்ந்தாலும்
வாலவார் முலைநல் லாளே வாதமுற் பவிக்குங்காயே

- பரராச சேகரகம்

Improper food schedule, intake of excessive water, sitting in the cold air during evening hours leads to Vatha diseases.

பாரினிற் பயப்பட்டாலும் பலருடன் கோபித்தாலும்
காரெனக் கருகியோடிக் கழுமரத்து ரத்தினாலும்
ஏர்பெறு தனது நெஞ்சின் மிகத்துக்க மடைந்திட்டாலும்
பாரிய காற்றினாலும் படரீனும் வாதங்காணும்

- பரராச சேகரகம்

Fear, angry, anxiety, stress, exposure to cool air cause Vatha diseases

6). According to Agasthiyar in Agasthiyar Gunavagadam,

விவரமடா அசதி சன்னி மூளை நோவு
விரிவான மூளையது மிருதுவாகி
அவனிதனில் திடமாகப் போவதாலும்
அப்பனே மூத்திர குண்டிக்காய் வியாதியாலும்
தவமுனிவர் தீர்க்காக்கை மேக ரோகம்
தன்மையுள்ள முத்தண்டு கொடிவியாதி
அவமிலாப் பரிச நரம்பழுத்தங் கண்டால்
அணுகுமடா வாதநோய் ஆகும்பாரே

- அகத்தியர் குணவாகடம்

Fatigue, Epilepsy, Brain diseases, renal diseases, Genito urinary diseases, and Connective tissue disorders induce Vatha diseases.

c). Factors of Kanmam (Hereditary):

In Siddha system many diseases are said to be caused by Kanmam which means the deeds committed by an individual in his / her present and previous births.

According to Agasthiyar Kanma Kandam-300,

நூலென்ற வாதம் வந்த வகைதானேது
துண்மையாய்க் கன்மத்தின் வகையைக் கேளு
காலிலே தோன்றியது கடுப்பதேது
கைகாலில் முழக்கியது வீக்கமது
கோலிலே படுகின்ற விருட்சமான
குழந்தை மரந்தனை வெட்டல் மேல்தோல்சீவல்
நூலிலே சீவஜந்து கால் முறித்தல்
நல்லகொம்பு தழைமுறித்தல் நலித்தல் தானே

- அகத்தியர் கன்மகாண்டம்

(பாடல் எண்.56, பக்கம் எண்.23)

Vatha Kanma Varalaru is concludedthat the psychological factors such as removing the bark of living trees, injuring the animals, cutting the branches in the living trees and plucking the leaves may produceVatha diseases.

மேலும்,

அந்தணர் கற்பு மாதர் அருளிய சாபத்தாலும்
முந்திய வினையாலும் முதிர்கர்ப்ப மேகத்தாலும்
சிந்தையிற் கொடுமையாலும் சிவகுரு நிந்தையாலுந்
தொந்தமாம் வியாதியாலும் தோன்றிடும் சூலைதானே

Soolai (Stabbing pain) may occur by the curse of noble men and women, due to evil deeds in previous birth, due to the genito urinary diseases produced by their parents, due to bad thoughts and curse of Guru.

CLINICAL FEATURES:

வழுத்தவே மூலாதாரத்தைப் பற்றியே
மருவியே மேலேறி முதுகுண்டாதல்
விழுத்தவே சிரசில் வந்து வியர்வுமாகி
விகுவாக நோவாகி மேனி கன்னி
பத்தவே உடம்பெங்கும் பஞ்சு போலாம்
பாங்கான மலசல மஞ்சளாகும்
குழுத்தவே தெண்டமாம் வாதந்தன்னைக்
கூறினோங் குணமெல்லாங் கூர்ந்து பாரே
கூர்ந்திட்ட மலசலங்கள் துரிதமானால்

கோண்ட டக்கிப் பின்புதான் கொடிதாய் தள்ளி
ஊர்ந்திட்ட சரீரத்தி லுதிர மீறி
ஊறத் தேய்த்து தலையதனி லெண்ணெய் வார்க்கில்
வார்த்திட்ட வழி நடக்கில் மெத்த வந்தான்
வாதந் தானுற்பவித்து நடை கொடாமல்
நார்ந்திட்ட நரம்போடு எலும்பிற் சூழ்ந்து
நணுகியே யோடி நெஞ்சி வேறுந் தானே

- யூகி வைத்திய சிந்தாமணி-800

(பாடல் எண்.288,289 பக்கம் எண்.109,110)

Siddha review of literature describedabout the diseases Thandaga Vatham.

i). **According to Thanvanthri Vaidhyam,**

வாயு வதிகமாய்ச் சிலேற்பனத்தைத்
தாமகட்டாகச் சேர்த்துத் தடித்திடுஞ் சரீரமெல்லாம்
நோமக் கட்டான மேனி நுவலிளைப் பெயர்ப்புத் தோன்றும்
தாமக் கட்டான ரோகந் தண்டக வாதமாமே

- தன்வந்திரி வைத்தியம்

Thanvanthri,explains that in Thandaga Vatham,Vatham associated withKapam results in generalised edema, obesity and generalised debility.

ii). **According to Roganiganirnayasaram:**

"தேகம் தண்டத்தை போல் விழுந்து அசைவில்லாமல் இருக்கும்"

Body is rendered like a log of wood.

iii). **According to Jeevaratchamirtham,**

வாயுவானது எண்ணெய் வஸ்து, மந்த வஸ்து, சீத வீரிய வஸ்து, தயிர், அதிக லவணம், பகல் நித்திரை, பதினான்கு வேகங்களை மறித்தல் ஆகிய இவைகளினால் பிறந்த சப்த தாதுக்களிலும் வியாபித்து அவைகளைக் கலைத்து விட்டு ராசயஸ்தானத்தை அனுசரித்துச் சிலேத்துமபித்தங்களைத் தன்னுடன் சேர்த்துக் கொண்டு அவயங்களின் செயலை மாற்றி விடும். இதனால் ரசாதி தாதுக்களில் மரத்தல் (Numbness), சீதளம் (Cold), உள்ளெரிச்சல் (Burning Sensation), சரீரங் கனத்தல் (Heaviness of body), ஞாபக மறதி (Loss of Memory), பிரமை (Psychosis), அதிக வேதனை (Severe pain), நீர்க்கட்டு (Anuria) என்னும் இக்குறிகுணங்களோடு தேகமானது தண்டத்தைப் போல விழுந்து அசைதலும், நீட்டலும், முக்கலும், எழுதலும் இல்லாதிருக்கும் (Body is rendered like a log wood and unable to flex, extend and rotate).

- அனுபோக வைத்திய தேவரகசியம்

(முதல் பாகம், பக்கம் எண்.164)

iii). **According to Sikitcharatna Deepam:**

வாயுவானது மூலாதாரத்தைப் பற்றி மேலேறி முதுகிலிருந்து சிரசில் வந்து வியர்த்து நோயுண்டாக்கி சர்வாங்கத்தையும் நோயுறச் செய்வதுடன் மலசலம் மஞ்சள் வர்ணமாகவும், தேகத்தை தண்டகம் போல் நீட்ட விடாமல் செய்யும்.

* Profuse sweating all over the body
* Yellow coloured urine
* Body is found to be like a log of wood

iv). **According to Panditharatna Dr. S.Chidambaranatha Pillai, Siddha Medical Literature Research Centre,**

Idupu Vatham has the characterised features of pricking pain and generalised weekness,pain in the lower back area,restricted spinal vertebrae motions.

இடுப்பது கடுத்து உளைந்து
இடைவிடா வலித்துக் கொள்ளும்
முடுக்கமாய் குனியவே தான்
முடுகியே நிமிர வொட்டாது
துடுக்கென வந்து அடரும்
சுரமது அற்பம் அற்பம்
சுடக்கென இடுப்பைச் சுற்றி
சார்ந்திடும் வாதம் தானே.....

- வாதநோய் மருத்துவம்
(பக்கம் எண்.114)

The Idupu Vatham**,** pain aggrevates while walking and subsides at rest; while lying on bed, the patient is unable to move their lower back.

மேலும்,

நடப்பென போது மெத்த நய்யவே வலிக்குமென்ன
கெடப்பெனபோதும் சற்றே குணமென தோன்றுமாகில்
படுப்பன போதும் யாமம் பாகியால் வாதமுண்டாம்
இடுப்பென சேரும் வாதத்தியலிது எண்ணுவீரே

- வாதநோய் மருத்துவம்
(பக்கம் எண்.114)

மேலும்

Thandaga Vatham, as described in Vatha Noi Maruthuvam , it was given below:

Due to cold food stuffs, day time sleep, in case of Thandaga Vatham the patient develops increased level of Vatham their by deranging the Pitham and Kapam.

This leads to burning sensation (உள் எரிச்சல்), psychosis (பிரமை) asthma (இளைப்பு) and severe pain (வேதனை).

In Thandaga Vatham, the low back bone gets inflammed resulting in generalised debility, pain, body becomes weaker and patient gets anger easily.

தண்டு வாதத்தின் குணத்தை சாற்றக்கோளாய் மடமாயிலே
பண்டேதண்டு மிக ஊதி பற்றிபொதுமி கொண்டிருக்கும்
விண்டோம் சில போதுளைவுண்டாம் மிகுந்த வாட்டமுண்டாம்
கொண்டே மனமும் தளர்ச்சியும் கோப மதிகம்காணுமென்றே

- வாதநோய் மருத்துவம்

(பக்கம் எண்.126)

Due to the changes in Uyir thathu specifically Vatham, the 96 thathuvangal gets affected. As said above, due to various causes the following changes occurs in Thandaga Vatham.

DIAGNOSIS IN SIDDHA:

A). **Piniyari Muraigal** (Methods of Diagnosis) is based upon three main topic namely,

- ❖ Poriyal Aridhal (Physical Examination, Perception)
- ❖ Pulanal Aridhal (Palpation)
- ❖ Vinadhal (Interrogation)

i). Poriyal Aridhal (Inspection):

'Poriyal Aridhal' means examining the five sense organs of perception.

ஞானேந்திரியங்களின் ஆய்வு		
புலன்கள்	**தொழில்கள்**	**தண்டக வாதத்தில் பாதிப்பு**
சேவி	ஒலிய அறிய செய்ய	இயல்பு
மெய்	உடலில் ஊற்றை அறிதல்	முதுகில் வலி, வீக்கம்
கண்	ஒளிய அறியச் செய்தல்	இயல்பு
நாக்கு	சுவையை அறியச் செய்தல்	இயல்பு
மூக்கு	வாசனையை நுகரச் செய்தல்	இயல்பு

கன்மேந்திரியங்களில் ஆய்வு		
புலன்கள்	**தொழில்கள்**	**தண்டக வாதத்தில் பாதிப்பு**
வாய்	பேசச் செய்யும்	இயல்பு
ஐக	இடுதலும், ஏற்றலும் செய்யும்	இயல்பு
கால்	நடக்கச் செய்யும்	கால்களில் வலி நடக்கச் சிரமம்
எருவாய்	மலத்தைக் கழிக்கும்	மலச்சிக்கல்
கருவாய்	கரு, சுக்கிலத்தைக் கழிக்கும்	இயல்பு

ii). Pulanal Aridhal (Palpation):

By examining the pulan i.e., the sense organ of the patient, the physician can able diagnose the disease.

The five senses are,

- Smell
- Taste
- Vision
- Sensation of touch
- Hearing

iii). Vinadhal (Interrogation):

Vinadhal is questioning and gathering information regarding the previous history of disease and clinical features which are much essential for diagnosis.

B). Envagai Thervugal (Eight Diagnostic Tools):

The excellent and unique method in the Siddha system is the Envagai Thervugal.

நாடி ஸ்பரிசம் நா நிறம் மொழி விழி
முலம் மூத்திரம் மருத்துவராயுதம்

- நோய் நாடல் நோய் முதல் நாடல்
முதல் பாகம் (பக்கம் எண்.270)

1. Naadi (Pulse):

Among the Envagai Thervugal, Naadi is most important. Naadi is felt as Vatham, Pitham and Kapam with the tip of the index, middle and ring fingers respectively over the lower end of the radius.

Normally Vatham, Pitham and Kapam are held in the ratio of 1:1/2:1/4. Derangement in this will reflect as disease. Naadi Nadai in Thandaga Vatham,

திருத்தமாம் வாதத் தோடே தீங்கொடு பித்தஞ் சேரில்
பொருத்து கள்தோறும் நொந்து போதவே பிடிக்கும் சூலை

- நோயின் சாரம் - சித்தமருத்துவம் (பொது)

(பக்கம் எண்.634)

காணப்பா வாத மீறில்
கால்கைகள் பொருத்து நோகும்

- காவியநாடி - சித்த மருத்துவம் (பொது)

(பக்கம் எண்.634)

சொல்லிய வையத்தோடு பித்தமுங் கூடிற்றானால்
வல்லியம் போலக் குத்தும் மைந்தனே எலும்பு தோறும்

- காவியநாடி - சித்த மருத்துவம் (பொது)

(பக்கம் எண்.634)

அறிந்துபார் வாதமே தனித்தானதால்
சரிந்திடவே கால் முடக்கும்

- அகத்தியர் ரத்தினச் சுருக்கம்

(பக்கம் எண்.634)

வாதத்தில் சேத்துமமாகில் வலியோடு வீக்கமுண்டாம்

- அகத்தியர் நாடி

In Thandaga Vatham the following Naadi Nadai are commonly felt.

- Vatham
- **Vatha** Pitham
- Pitha **Vatham**

2. Sparisam (Sensation to touch):

Through Sparisam, heat or cold, smoothness, roughness, sweat, dryness of skin, patches, swelling, abnormal growth, ulcer, pain can be felt. In Thandaga Vatham, heat, swelling, pain is found in some cases.

3. Naa (Tongue):

In Thandaga Vatham, no abnormality is seen in Naa.

4. Niram (Colour):

In Thandaga Vatham, some skin colour changes seen in affected area due to inflammatory mechanism.

5. Mozhi (Voice):

In Thandaga Vatham, no abnormality is seen.

6. Vizhi (Eyes):

In Thandaga Vatham, no abnormality seen in vizhi.

7. Malam (Faeces):

In Thandaga Vatham, Constipation is reported in some cases.

8. Moothiram (Urine):

In urine, Neerkuri and Neikuri examinations are done.

Neikuri (Oil Examination):

The observation the spreading oil pattern as follows:

அரவென நீண்டினஅ.ஃதே வாதம்
ஆழிபோற் பரவின் அ.ஃதே பித்தம்
முத்தொத்து நிற்கின் மொழிவ தென் கபமே

- நோய் நாடல் பாகம்-1

(பக்கம் எண்.298, 299)

Neerkuri:

வந்த நீர்க்கரி யெடை மணம் நுரை எஞ்சலென்
றைந்தியலுளவை யறைகுது முறையே

- சித்த மருத்துவாங்க சுருக்கம்

(பக்கம் எண்.510)

In urine examination the following characteristic features are observed namely.

Niram	-	Colour
Edai	-	Specific gravity
Manam	-	Smell
Nurai	-	Frothy nature
Enjal	-	Quality of urine voided

Apart from these, frequency of micturition, abnormal constituents such as sugar, protein, blood stains, pus, crystals also to be found out.

In Thandaga Vatham, straw or hay coloured urine was noticed in Neerkuri.

C). Paruvakaalam (Seasonal variations):

Sl. No.	State of Kuttram	Kaalam
1.	Vatham thannilai adaithal	Munpani Kaalam, Pinpani Kaalam, Koothirkaalam, Elavenil Kaalam
2.	Vatham thannilai valarchi	Muthuvenil Kaalam
3.	Vatham vetrunilai valarchi	Karkaalam

முதுவேனிற் காலத்தில் நமது உடலில் வறட்சி ஏற்பட்டு வளிநோய் வருவதற்கு ஏதுவாகிறது.

D). திணை (Geographical distribution):

குறிஞ்சி : மலையும், மலை சார்ந்த பகுதியும்

முல்லை : காடும், காடு சார்ந்த பகுதியும்

மருதம் : வயலும், வயல் சார்ந்த பகுதியும்

நெய்தல் : கடலும், கடல் சார்ந்த பகுதியும்

பாலை : மணலும், மணல் சார்ந்த பகுதியும்

முல்லை மற்றும் நெய்தல் நிலங்களில் வாத நோய்கள் பெருமளவில் ஏற்படும்.

E). ஏழு உடல் தாதுக்களின் ஆய்வு (Seven Udal thathukkal Examination):

Sl. No.	Udal Thathukkal	Increased conditions	Decreased conditions
1.	Saaram	Loss of appetite, excessive salivation.	Tiredness, fatigue, diminished activity of the sense organs.
2.	Senneer	Boils and tumours in different parts of the body, spleenomegaly, colic pain, increased blood pressure, red eyes and skin, Jaundice, leprosy, Haematuria.	Tiredness, Lassitude, Anemia.
3.	Oon	Tumours or extra growth around the neck, face, abdomen, thigh, genitalia etc., with dyspnoea.	Muscle wasting

4.	Kozhuppu	Tumours or extra growth around the neck, face abdomen, thigh, genitalia etc., with dyspnoea and loss of activity.	Pain
5.	Enbu	Extra growth of bone and teeth	Weak bones, teeth, nails and hair.
6.	Moolai	Heaviness, swollen eyes, swollen phalanges, oliguria and non-healing ulcers.	Osteoporotic changes.
7.	Sukkilam or Suronitham	Increased sexual activity and symptoms as that of urinary calculi.	Infertility, pain in genitalia.

In Thandaga Vatham:

Saaram, Seener, Kozhuppu, Enbu thathukkal are commonly affected.

Saaram : Weakness, tiredness of body.

Seener : Early morning stiffness occurs in affected joints.

Kozhuppu : Restricted movements in joints and reduced intervertebral disc.

Enbu : Produces degeneration in lumbar vertebrae, spondylotic changes and extra osteophytic formation

F). முக்குற்றங்களின் ஆய்வு (Mukkutram examination):

Sl. No.	Vatham	Location	Function	In Thandaga Vatham
1.	Pranam	Chest region	Regulate respiration and controls the mental functions, functions of heart, lungs and brain	Not Affected
2.	Abanam	Pelvic region	Control excretion such as sweating evacuation of stools, ejaculation of sperms, micturition, menstruation and parturition.	May be Affected

3.	Viyanan	Nose and skull	Helps in various movements of the body and responsible for nervous functions and Sensation	Affected
4.	Uthanan	Thorat	Responsible for speech, vomiting hiccough , cough.	Not affected
5.	Samanan	Navel	Regulates the digestion and controls all the other vayus.	Affected
6.	Nagan	Eyes	Helps in opening and closing of the eyes, intelligence.	Not affected
7.	Koorman	Eye	Responsible for vision, closure of eyelids.	Not affected
8.	Kirukaran	Saliva	Secretion of saliva and mucous secretion in nasal cavity, helps concentration.	May be affected
9.	Devathathan	Ocular muscles	It is responsible for laziness and eyeball movements	May be affected
10.	Thananjeyan	-	It is responsible for degradation of body after death	-

Pitham:

Sl. No.	**Pitham**	**Functions**	**In Thandaga Vatham**
1.	Analagam	Digestion	May be affected
2.	Ranjagam	Gives nutrition to blood	May be affected
3.	Sathagam	Responsible for wilful activities	Affected
4.	Prasagam	Gives luster to skin	Not affected
5.	Alosagam	Gives strength to eyes	Not affected

Kapam:

Sl. No.	Kapam	Functions	In Thandaga Vatham
1.	Avalambagam	Controls other Kapam	Not affected
2.	Klethagam	It lubricates the food	May be affected
3.	Pothagam	Responsible for taste sensation	Not affected
4.	Tharpagam	It acts as coolent for eyes	Not affected
5.	Santhigam	It maintains the integrity of joints	Affected

DIFFERENTIAL DIAGNOSIS:

Thandaga Vatham is differential from the other diseases,

1. ஆசுவதம்ப வாதம்:

வாதமா யுடல்வெளுத்து வழவெல் லாநேரம்
மயக்கமோ டிருமலா யூளை யுண்டாம்
நேதமாய் நெஞ்சடைத்தப் பொறி கலங்கும்
நெருப்பாக உடல்காணு நெடுமூச்சுண்டாம்
கோதுதான் மயக்கத்தில் மருந்தி நீட்டால்
குளிர்ச்சியாய்க் கோபிக்குங் கூச்சலுண்டாம்
பாதந்தான் திமிருண்டாய் முட்போலாகும்
படுத்த ஆசுவதம்பம் பகரலாமே

- யூகி வைத்திய சிந்தாமணி-800

(பாடல் எண்.284, பக்கம் எண்.108)

The clinical features are:

- Paleness of the body
- Cough
- Heaviness of chest
- Numbness of both lower limbs
- Pain present in spino-vertebral column

2. ஊருத்தம்ப வாதம்:

ஆமென்ற வாதமது உள்ள டங்கி
ஆடித்துடைதான் குறங்கிரண்டு மளவாய்ப் பற்றி
காமெனற் கைகாலில் விரலு சுற்றிக்
கனத்துமே சாணியது பொதிந்தார் போலத்

தேமென்ற சிரந்தனிலே பார முண்டாய்த்
தேமெங்கு மூதியே திமிருண்டாகும்
நாமென்ற நடக்கொணர வொடுக்க மாகி
நலியூருந் தம்பமது நனுகுங்கானே

- யூகி வைத்திய சிந்தாமணி-800

(பாடல் எண்.260, பக்கம் எண்.98)

The clinical features are,

- Heaviness in both thighs.
- Feelings of cow dung applied over fingers of both hands and feet (Paraesthesia or Hypoaesthesia).
- Whole body perceives numbness.
- Difficulty in walking.

3. வாதஸ்தம்பம்:

உற்பவிக்கும் வாதமது எழுந்து பொங்கி
உயர்காலின் புறவடியைக் குடைந்து பற்றி
தெற்பவிக்கும் வீக்கமாய்ச் செழும்ப லுண்டாய்த்
தேகமெங்கும் நோவாகித் திமிரு மாகி
விற்புவிக்கும் வில்லுபோல விதனமாகி
மிடுக்கான மாந்தனைப் போல் விதனமாகி
பற்பவிக்கும் பரன்றனையே நினையாமூர்
படுகின்ற வாதஸ்தம் பமுமாம் பாரே

- யூகி வைத்திய சிந்தாமணி-800

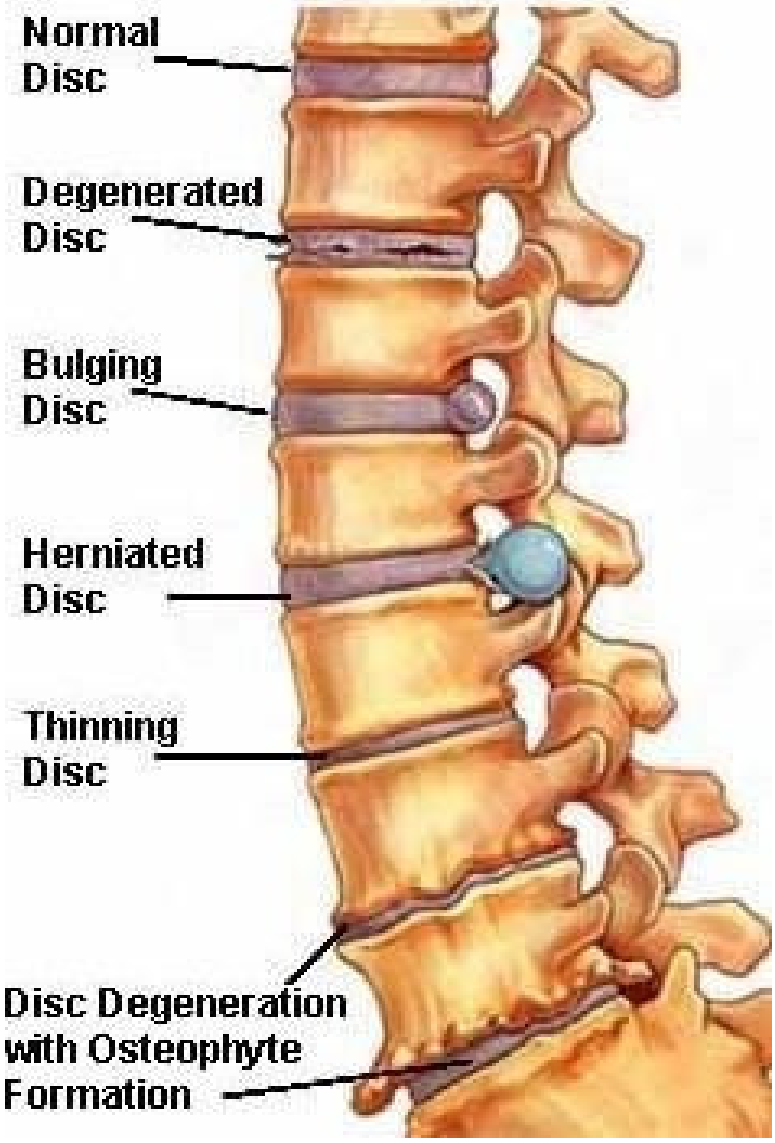
(பாடல் எண்.254, பக்கம் எண்.96)

The clinical features are,

- ❖ Dorsum of the feet gets affected (L5-S1), they look shining with edema, intense pain.
- ❖ Whole body appears to be weak andfatigue.
- ❖ Body is bend like abow.
- ❖ Makes a strong built man to bend and bow whilewalking.
- ❖ In Thandaga Vatham, diffuse low back pain, stiffness, radiating pain to lower limbs (Radiculopathy), yellow coloured stools and urine are present.

3.4 MODERN ASPECT

LUMBAR SPONDYLOSIS



LUMBAR SPONDYLOSIS;

Lumbar Spondylosis is a form of lower back pain and is an important clinical, social, economic and public health problem affecting the worldwide population. It is a disorder with many possible etiologies and many definitions.

DEFINITION:

Lumbar spondylosis changes in the spine are frequently referred to as osteoarthritis. For example, the phrase spondylosis of the lumbar spine means degenerative changes such as osteoarthritis of the vertebral joints and degenerating intervertebral dicss(degenerative disc disease) in the low back.

TACKLING THE TERMINOLOGY:

The terms lumbar osteoarthritis, disk degeneration, degenerative disk disease, and spondylosis are used in the literature to describe anatomical changes to the vertebral bodies and intervertebral disc spaces that may be associated with clinical pain syndromes. Within the literature, lumbar spondylosis encompasses numerous associated pathologies including spinal stenosis, degenerative spondylolisthesis, osteoarthritis, spinal herniation and many others.

EPIDEMIOLOGY:

Age:

The incidence of lumbar spondylosis is 27-37% of the asymptomatic lower back pain population. Worldwide, more than 80% of individuals older than 40 years have lumbar spondylosis, increasing from 3% of individuals aged 20-29 years. Approximately 84% of men and 74% of women have vertebral osteophytes, most frequently at T9-10 and L3 levels. Approximately 30% of men and 28% of women aged 55-64 years have lumbar osteophytes. Approximately 20% of men and 22% of women aged 45-64 years have lumbar osteophytes.

Sex:

Sex ratio reports have been variable but are essentially equal. Gender seems to be distinctly in the form of lumbar spondylosis, and disc space narrowing with or without osteophytes in women may be a risk factor for low back pain.

LOCATION:

Lumbar vertebrae is most frequently affected at the level of L4-S1 level.

AETIOLOGY:

Aetiology for this disorder was explained below

Degenerative Causes:

There are primary and secondary causes

a) Primary causes:

- Genetic Factor
- Manaul Labour
- Metabolic Factor
- Senility
- Vicious attitude

b) Secondary causes

- Osteoarthritis
- Rheumatoid arthritis
- Metastatic carcinoma
- Lymphoma of Spine
- TB Spine
- Road accidents
- Accidental Injury
- Old Lumbar Fracture
- Past spine surgery
- Acquired narrowing of lumbar spinal canal stenosis

c) Occupational causes

d)Hereditary Factors.

Congenital narrowing of the cervical spinal canal (myelopathy is often seen when canals sagittal diameter is 12mm or less)

Segmental defect-Hemi vertebra,Fushed vertebra.

e) Past spine surgery

f) Obesity

g) Smoking-decrease water content in disc

1) Acquired narrowing of lumbar spinal canal due to

- Osteophytes
- Sacralisation of L5 vertebrae
- Ossified Posterior Longitudinal Ligament(OPLL)
- Facet joint hypertrophy (results foraminal stenosis & compression of root of radiculararteryy)
- Hypertrophied ligamentum flavum (Compress the cord during extension)

PATHOPHYSIOLOGY:

The high incidence of simultaneous degenerative changes to the intervertebral disc, vertebral body, and associated joints suggests a progressive and dynamic mechanism, with interdependent changes occurring secondary to disc space narrowing.

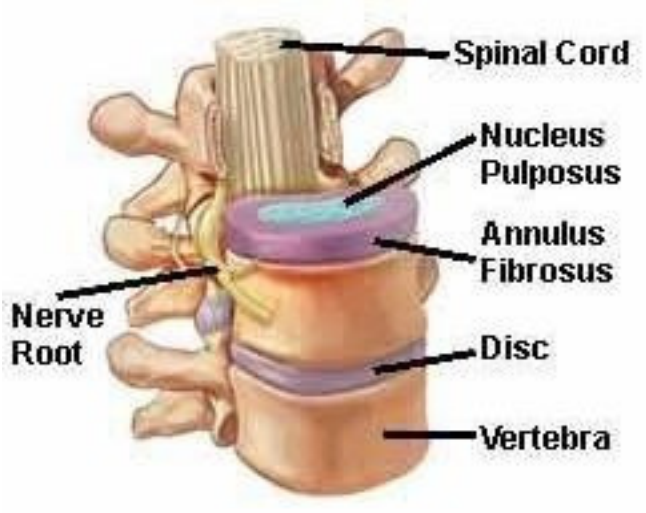
Intervertebral Joints:

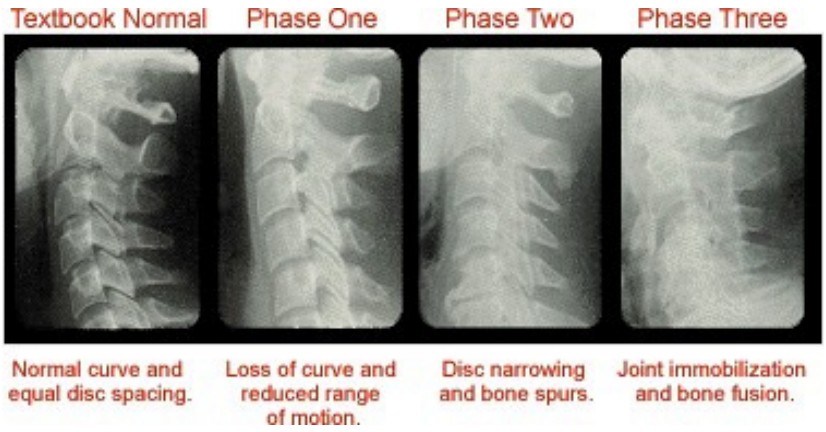
Adjoining vertebrae are connected to each other at three joints. There is a median joint between the vertebral bodies and two joints right and left between the articular processes.The joints between the articular processes are plane synovial joints.The joint between the vertebral bodies is a symphysis. The surfaces of the vertebral bodies are lined by thin layers of hyaline cartilage. Between these layers of hyaline cartilage there is a thick plate of fibrocartilage which is called the intervertebral disc.

PATHOPHYSIOLOGY

	FACET JOINTS	CLINICAL PRESENTATION	INTERVERTEBRAL DISC
PHASE I DYSFUNCTION	Synovitis Minimal cartilage degeneration	Restricted movement Unilateral radicular symptoms	Circumferential and radial annular tears
PHASE II UNSTABLE	Joint capsule laxity Facet joint subluxation Subperiosteal osteophytes	Increased movement Unilateral radicular symptoms	• Tears all the way through the annulus • Complete internal disc disruption • Circumferential annular bulging • Loss of disc height
PHASE III STABILIZATION	Periarticular fibrosis Osteophytes	Restricted movement Multilevel bilateral radicular symptoms	Ossification

Adapted from Kirkaldy-Willis W, Gerhart T. Managing Low Back Pain, ed 4. New York, 1991, Churchill Livingstone.





Intervertebral Discs:

These are fibro cartilaginous discs which intervene between the bodies ofadjacent vertebrae and bind them together. Their shape corresponds to that of the vertebral bodies between which they are placed. The thickness of the disc varies in different regions of the vertebral column, and in different parts of the same disc. In the cervical and lumbar regions the discs are thicker in front than behind, while in the thoracic region they are of uniform thickness. The discs are thinnest in the upper thoracic region and thickest in the lumbarregion.

The discs contribute about one-fifth of the length of the vertebral column. The contribution is greater in the cervical and lumbar regions than in the thoracic region.

Each disc is made up of the following two parts:

1. Nucleus Pulposus is the central part of the disc. It is soft and gelatinous at birth. It is kept under tension and acts as a hydraulic shock absorber. With advancing age the elasticity of the disc is muchreduced.
2. Annulus Fibrosus forms the peripheral part of the disc. It is made up of a narrower outer zone of collagenous fibres and a wider inner zone of fibrocartilage. The fibres from laminae that are arranged in the form of incomplete rings. The rings are connected by strong fibrous bands. The outer collagenous fibres blend with the anterior and posterior longitudinal ligaments.Intervertebral discs are believed to undergo a "degenerative cascade" of three overlapping phases that may occur over the course of decades.

Phase-I (Dysfunction Phase) 15 to 45 years describes the initial effects of repetitive microtrauma with the development of circumferential painful tears of the outer, innervated annulus, and associated end-plate separation that may compromise disk nutritional supply and waste removal. Such tears may coalesce to become radial tears, more prone to protrusion, and impact the disc□s capacity to maintain water, resulting in desiccation and reduced disk height. Fissures may become ingrown by vascular tissue and nerve endings, increasing innervation and the disc□s capacity for pain signal transmission.

Phase-II (Instability Phase) 35 to 70 years is characterized by the loss of mechanical integrity, with progressive disc changes of resorption, internal disruption, and additional annular tears, combined with further facet degeneration that may induce subluxation andinstability.

Phase-III (Stabilization Phase) 60 years and above is continued disc space narrowing and fibrosis occurs along with the formation of osteophytes and transdiscal bridging.

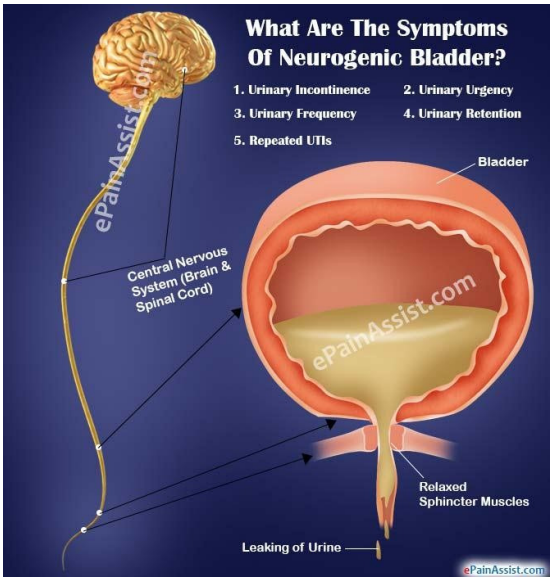
There is a spectrum of pathological changes in facial joints and the disc and the interaction of these changes. Adjacent pedicles approximate with a narrowing of the superior-inferior dimension of the intervertebral canal. Laxity due to modest redundancy of the longitudinal ligaments enables bulging of the ligamentum flavum and potential for spine instability. Increased spine movement permits subluxation of the Superior Articular Process (SAP), causing a narrowed anteroposterior dimension of the intervertebral and upper nerve root canals. Laxity may also translate into altered

weight mechanisms and pressure relationships on vertebral bone and joint spaces believed to influence osteophyte formation and facet hypertrophy to both inferior and superior articular processes with risks for projection into the intervertebral canal and central canal, respectively. Oblique orientations of the articular processes may further cause retrospondylolisthesis, with resulting anterior encroachment of the spinal canal, nerve root canal, and intervertebralcanal.

Biochemical research exploring osteophyte formation supports the above process. Osteophyte lipping is believed to form at periosteum through the proliferation of peripheral articular cartilage which subsequently undergoes endochondral calcification and ossification. Changing weight mechanics and pressure forces as well as alterations in oxygen tension and dynamic fluid pressure appear to be influential factors in osteophyte formation. Mesenchymal stem cells of the synovium or periostium are likely precursors, with synovial macrophages and a milieu of growth factors and extracellular matrix molecules acting as probable mediators in this process.

SYMPTOMS


PAIN


What Are The Symptoms Of Neurogenic Bladder?
1. Urinary Incontinence
2. Urinary Urgency
3. Urinary Frequency
4. Urinary Retention
5. Repeated UTIs
Bladder
Central Nervous System (Brain & Spinal Cord)
Relaxed Sphincter Muscles
Leaking of Urine
ePainAssist.com


Loss of balance


Burning Back Pain


NUMBNESS OR
TINGLING?

SIGNS AND SYMPTOMS:

When a patient suffers from lumbar spondylosis, it is possible that osteophytes are formed. These osteophytes are bony overgrowths that occur due to the stripping of the periost from the vertebral body.

- Pain- can be produced when a neural foraminal stenosis is formed, which comes from the formation ofosteophytes.
- Joint stiffness, which can limitmotion.
- Neurologic claudication, whichincludes:
- Lower backpain,
- Legpain,
- Numbness when standing andwalking.
- Radiating pain towards the lowerextremities
- Diffuse tenderness in lumbarbone
- Exacerbation of painon movements
- Pain increased on forward bending, sneezing,coughing
- Paraesthesiaandsensorylossonaffectedarea
- Burning and tingling sensation in lowerlimb
- Painandstiffnessinlowback inthemorninghours

Less Common Symptoms:

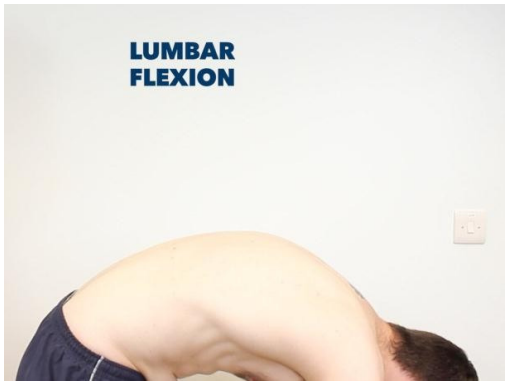
- Loss ofbalance
- Neurogenicbladder.

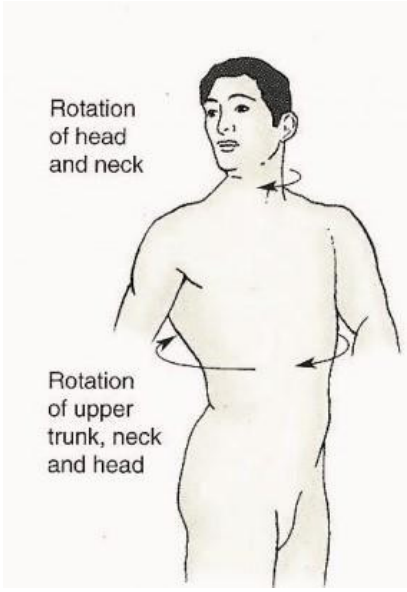
EXAMINATION:

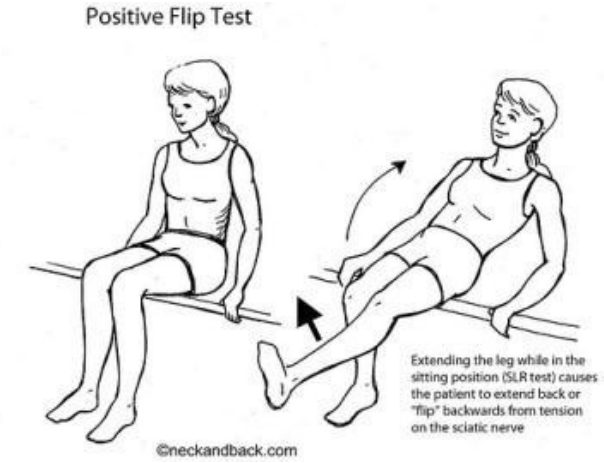
When a physician performs an examination for lumbar spondylosis, it is advised to follow the principles of the general spine examination and apply them to this specific pathology. General examination of the spine:

- The examination should begin with careful observation during the whole consultation.
- It is essential to observe the patient's gait andposture.

EXAMINATION OF LUMBAR SPINE







1)Inspection:

- Inspection of the entirespine.
- Look for any obvious swellings or surgicalscars.
- Assess for deformity: scoliosis, kyphosis, loss of lumbar lordosis or hyperlordosis of the lumbar spine. Look for shoulder asymmetry and pelvic tilt.

2)Palpation:

- Palpate for tenderness over bone and softtissues.
- Perform an abdominal examination to identify any masses and consider a rectal examination to exclude other pathologies in thisregion

Inference:

- No tenderness to palpation is noted, but some discomfort can be elicited with deep percussion over the midline of the lumbararea.
- Physical findings that may also be present include antalgic or normal gait, tight lumbar musculature and hamstrings, hyperlordosis, and buttock or thigh pain.

1)Movement:

- Examination of the spine must also include examination of the shoulders and examination of the hips to exclude these joints as a cause of thesymptoms.

To test flexion:

- Instruct the patient to bend forwards as much as possible at the waist.
- Normal flexion is 80^{o} or finger tips 3-4 inches from the floor.

Lateral Flexion:

- Instruct the patient to bend to the leftand to the right as far as possible.
- Normal range is 35^{o} on each side.

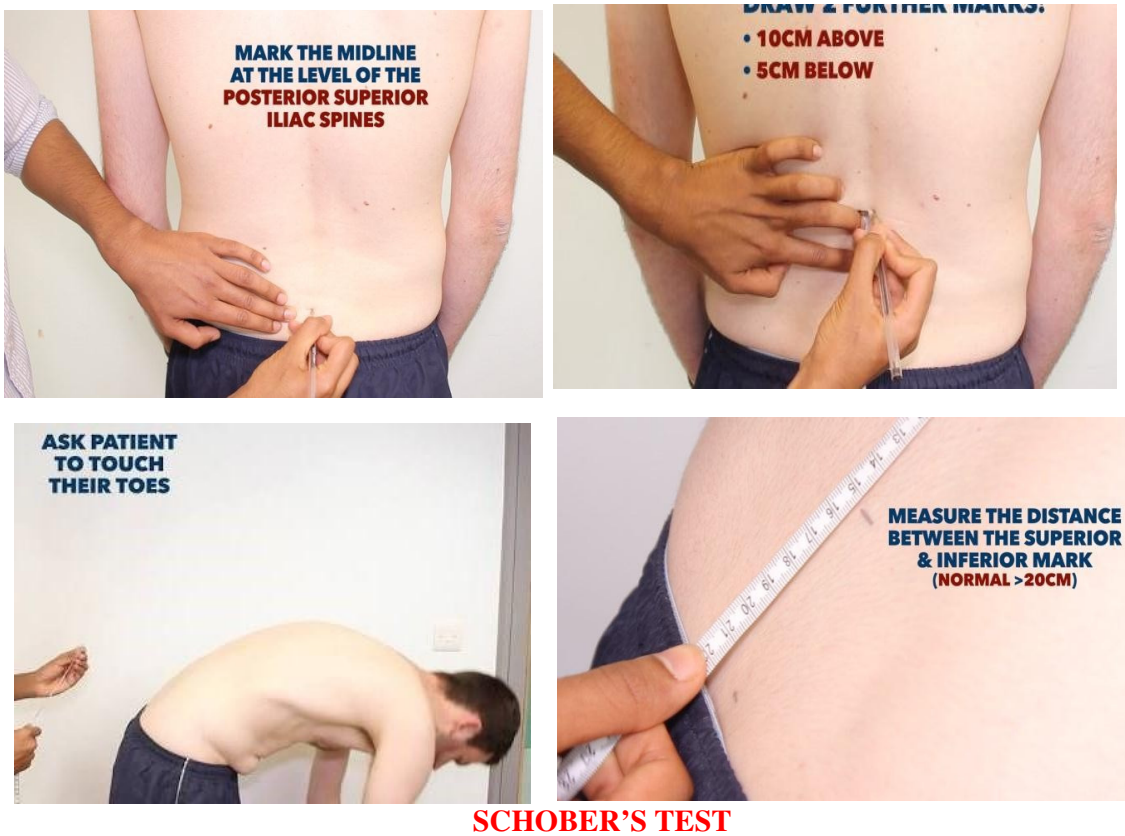
Extension:

- Instructs the patient to bend at waist as far backward as possible.
- Normal range is20^{o}-30^{o}.

Rotation:

- Instructs the patient to rotate from the waist to the left and to the right as far as possible. Normal range is 45oC possible

FEMORAL NERVE STRETCH TEST



SCHOBER'S TEST

1. Neuro vascular examination:

- A thorough examination of sensation, tone, power and reflexes should be performed.
- Always consider the possibility of acute spinal cord compression, which is a neurosurgicalemergency.
- All peripheral pulses should also be checked, as vascular claudication in the upper and lower limbs can mimic symptoms of radiculopathy or canalstenosis

2. Tests for Examination:

- Straight Leg Raising test(SLR)
- Braggards test
- Femoral nerve stretch test
- Schobers test
- Forward bending to touch the toes
- Flip test
- Lassegue test
- Bowstring sign

OUTCOME MEASURES:

Numeric Pain Rating Scale (NPRS): The patient is asked to score 3 pain rating, worse / current / best over the last 24 hour. The score for this scale is the average of these 3 values. This scale is a variant of the VAS but also assess pain intensity.

Roland Morris Disability Questionnaire (RMDQ): This questionnaire contains sentences that people have used to describe themselves when they have back pain on that specific day. As people read the list they might recognize themselves and then they must tick that box. A score is appointed according to the amount of boxes the patient fills in. This questionnaire makes it possible to follow changes in time.

Oswestry Disability Index (ODI): This index is made to evaluate how back pain invalidates people in their daily activities (sleeping, self-care, sex life, social life and travelling). Each question contains 6 categories (0: no limitation 6: most limitation). The score is calculated by the sum of the 10 questions, multiplied by 2. This value represents the percentage of invalidation.

Pain Self-Efficacy Questionnaire (PSEQ): This questionnaire rates how confident patients feel performing activities despite the pain. This is indicated on a

scale from 0 (no confidence) to 6 (completely confident). Al the scores are then added up to a score from 0 to 60. Where the closer to 60 means that the patients have a stronger self-efficacy belief. There are also short versions of this questionnaire available who shows also a great responsiveness.

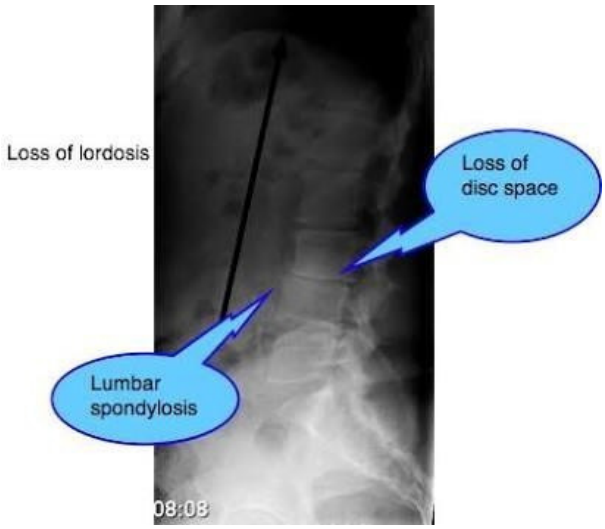
Patient-Specific Functional Scale: Questionnaire where patients are asked to identify up to three activities that they had difficulty with or are unable to perform as a result of their back pain. Each item is given a score of 0-10 (unable- able). The total score is assessed by the sum of the activity scores/number of activities (Minimum detectable change (90%CI) for average score = 2 points, Minimum detectable change (90%CI) for single activity score = 3 points) of all those questionnaires the NRPS is recommended for assessing pain because of this ease of administration and responsiveness. The ODI and RMDQ are recommended for assessingfunctioning.

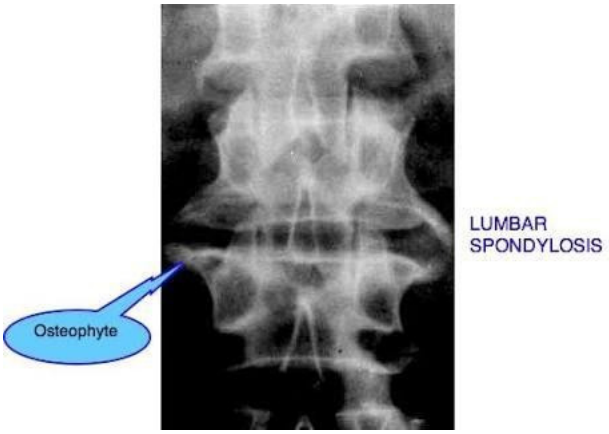
DIAGNOSIS:

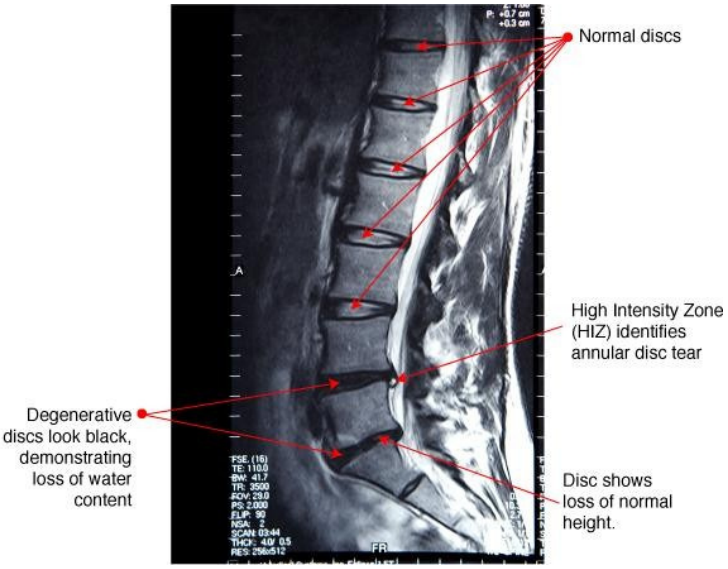
For the clinical diagnosis of lumbar spondylosis, a thorough investigation is necessary to ensure that other pathologies are excluded.

- **MRI:** Shows the greatest details in the spine and is used to visualize the intervertebral discs, including the degree of disc herniation, if present. An MRI is also used to visualize the vertebrae, the facet joints, the nerves, and the ligaments in the spine and can reliably diagnose a pinchednerve.
- **X-Rays:** show bone spurs on vertebral bodies in the spine, thickening of facet joints (the joints that connect the vertebrae to each other), and narrowing of the intervertebral discspaces.
- **CT scan**: able to visualize the spine in greater detail and can diagnose narrowing of the spinal canal (spinal stenosis) whenpresent.
- **SPECT**: Single-photon emission computedtomography.
- Bone scintigraphy is used to further evaluate patients with suspected spondylosis. Controversy surrounds the designation of one of these tests as most useful in the evaluation ofspondylosis.

RADIOLOGICAL FINDINGS







Myelogram:

Helping in detecting:

- Intra spinallesion
- Spinalstenosis
- Any compression of the spinalcord
- **Discography**
- **Nerve conduction studies**
- **EMG**

These procedures were validated in several studies, which concluded that MRI was effective (92% sensitivity) in identifying pars lesions. CT scan was also used as a diagnostic procedure, but the result weren□t equally positive. That is the reason why MRI is advised as the best method of diagnosis.

DIFFERENTIAL DIAGNOSIS:

1. Osteoporosis
2. Multiple myeloma
3. Multiple sclerosis
4. Extra dural tumor
5. Ankylosis spondylosis
6. Spino vascular insufficiency
7. Peripheral neuropathy
8. Herpes zoster
9. TB Spine
10. Rheumatoid arthiritis
11. SLE
12. Mixed connective tissue disorder

COMPLICATIONS:

- Nerve compression from posterior osteophytes is a possible complication only if a neuroforamen is reduced to less than 30% of normal.
- If lumbar spondylosis projects into the spinal canal, spinal stenosis is a possible complication
- An isolated report of a bony L4 mass pressing on the duodenum has been described.
- Cauda equina syndrome
- Neurogenic claudication
- Paraplegia
- Conus Medullaris Syndrome

CHAPTER-IV

MATERIALS AND METHODS

A Prospective open labelled non randomised Phase-II Clinical trial to assess the therapeutic efficacy of the siddha formulation Munnai Ilai Kudineer for the treatment of THANDAGA VATHAM (Lumbar Spondylosis) is carried out in Post Graduate Department of Pothu Maruthuvam, Government Siddha Medical College and Hospital, Palayamkottai.

Selection of the Cases:

Totally 40 cases were selected. Out of 40, 20 cases were selected as Inpatients and 20 cases treated as Out patients. These cases were selected from the Out patient Department of Pothu Maruthuvam according to the inclusion and exclusion criteria.

Aetiological factors:

The seasonal variations and precipitating factors like emotional stress and strain, trauma, occupation and food habits were enquired and recorded. The socio economic status, family history and other significant diseases treated already, were thoroughly registered.

1. INCLUSION CRITERIA

Patients having textual symptoms of Lumbar Spondylosis will be taken as a subject to the study

- Patients of either sex were included in the study
- Patients of age group between 30-60 yrs.
- Patients with low back pain with radiograph of lumbosacral spine showing,
- Loss of natural lumbar lordosis
- Reduction in disc space
- Formation of osteophytes
- Osteoporosis
- Subluxation of one vertebra over another.

2. EXCLUSION CRITERIA

- Age below 30 years and above 60 years.
- Diabetes mellitus
- Auto immune diseases like SLE
- Chronic Kidney Disease
- Fracture of Spine

- Tuberculosis of Spine
- Congenital spino vertebral deformities
- Cardiac disease.

3. WITHDRAWAL CRITERIA

The patients were withdrawn from the trial if,

- Occurrence of adverse effects with given treatment, also if patient needs emergency management.
- The investigator feels that the protocol has been violated.
- Further continuation of the study is likely to be detrimental to health of the patients
- The patient is not willing to continue the trial

Diagnosis:

The diagnosis is made by following Siddha diagnosis methods:

- Poriyalaridhal
- Pulanalaridhal
- Vinadhal
- Envagai thervugal
- Udal thathukkal
- Kaalam
- Nilam

Haematological Investigations:

- HB%
- Total WBCCount
- DifferentialCount
- Erythrocyte SedimentationRate
- Blood SugarR/PP/F
- BloodUrea
- SerumCholesterol
- SerumCreatinine
- Serum UricAcid

Urine analysis:

- Albumin
- Sugar
- Deposits

Specific Investigations:

- RAfactor
- ASOtitre
- CRP

Radiological Investigations:

- X-Ray of Lumbar Spine (AP and lateralview)

Assessment of result:

The results were assessed on the basis of symptomatic relief, improvement in grade of cardinal signs and range of movements and by back pain functional assessment scale.

The difference in the grade and score before and after treatment represents the improvement in the treatment.

Further the biochemical, pharmacological and acute toxicity studies were done in Government Siddha Medical College, Palayamkottai and KM Pharma College Madurai.

Treatment:

The clinical trial drug **"MUNNAI ILAI KUDINEER"**30ml twice a day after food is given for 30 days till the end of the course.All the patients admitted for the study were given uniformly regular hospitaldiet.After discharge all the patients were advised to attend the Out patient Department of Pothu Maruthuvam, Government Siddha Medical College and Hospital, Palayamkottai for further followup.

Ethical Review

The study was conducted in accordance with the ethical principles that are consistent with good clinical practice guidelines and obtained prior approvals before start of the teial from the Institunal Ehical Committee of GSMCH ,palayamkottai(GSMC-IV-IEC/2017/Br-I/07/29.05.2017) and Instiuional animal ethical committee (IEAC) of K.M College of pharmacy, Madurai (TNMGRMU/KMCP/IEAC/20/2018). The trial was applied and approved by the Clinical Trial Registry of India.

CHAPTER-V

OBSERVATION AND RESULT

5.1 PRE CLINICAL STUDY

5.1.1ANALGESIC ACTIVIY

Evaluation of Analgesic Activity of Siddha formulation Munnai Ilai Kudineerin Animal Models

Introduction

Inflammation and pain are common nonspecific manifestations of many diseases. Although non-steroidal anti-inflammatory drugs (NSAIDs) and opiates have been used classically in these conditions, but some adverse reactions occur with these drugs such as gastrointestinal disturbances, renal damage, respiratory depression, and possible dependence (1-2). In recent years, there has been an increasing interest to find new anti-inflammatory and analgesic drugs with possibly fewer side effects from natural sources and siddha formulations.

Based on Indian folk medicine, in the present study analgesic effects were evaluated for siddha formulation Munnai Ilai Kudineer in mice using acetic-acid writhing test..

Animals

24 adult male albino mice (25-35 g) were housed in animal house,K.M.College of Pharmacy,Madurai,under standard laboratory conditions (temperature 23 ± 2 ˚C) with 12 h dark and 12 h light cycle. The animals had free access to standard dry pellet diet and tap water ad libitum

Analgesic activity

Acetic acid-induced writhing test

The acetic-acid writhing test was performed using the reported procedure (3)

Groups of rats (n=6), were administered with100 and 200 mg/Kg of siddha formulation Munnai Ilai Kudineer,10 mg/Kg Diclofenac as positive control group and 1 mL distilled water as negative control group. After 30 minutes the animals were administered with i.p. injection of 0.1 mL acetic acid (0.6%). Then the count of abdominal contractions of animals during 30 minutes after acetic acid injection was reported and the Percentage Analgesic Activity (PAA) was calculated by using the following formula:

PAA = ((C- CD)/CD) ×100

C = Mean of contractions' count in animals treated with different doses of siddha formulation Munnai Ilai Kudineer and Diclofenac sodium

CD = Mean of contractions' count in animals served as negative control

Statistical analysis

The results are reported as mean ± S.E.M. The statistical analyses were performed using one way analysis of variance (ANOVA). Group differences were calculated by post hoc analysis using Tukey's test. For all tests, differences with values of $P<0.05$ were considered significant.

Results

Acetic acid-induced writhing response

The second study showed that the application of different doses of siddha formulation Munnai Ilai Kudineer had significant analgesic effects in the animals under investigation. The results of doses 100 and 200 mg/Kg were significant and comparable with the effect of Diclofenac sodium in analgesic activity (Table 1).

Table 5.1.2.a. Effects of siddha formulation Munnai Ilai Kudineeron acetic acid–induced writhing response (N=6 in each group).

Groups	**Treatment**	**(number of writhing movements) (Mean ± S.E)**	**Percentage %**
Group I	Distilled water	30.00 ± 2.52	
Group II	Diclofenac sodium 10mg/kg	6.30 ± 0.98*b	79.00%
Group III	100mg/kg Munnai Ilai Kudineer	15.45 ± 1.72*b	48.50%
Group IV	200mg/kg Munnai Ilai Kudineer	13.40 ± 1.32*b	55.33%

- Values are expressed as mean **±** SEM.

* (b) Values are significantly different from Toxic control G2 at $P<0.01$.

Discussion and conclusion

The analgesic activity was assessed by writhing test which has been reported to be useful for investigation of peripheral antinociceptive activity and performed as a chemical pain model (4,5). The siddha formulation Munnai Ilai Kudineerdemonstrated a dose-dependent, significant antinociceptive activity in animal models of pain. Acetic acid believed to increase the PGE2 and PGF2α in peritoneal fluid (6). The analgesic activity shown in models of pain is indicative that siddha formulation Munnai Ilai Kudineer might possess centrally and peripherally mediated antinociceptive properties.

Chemical components of siddha formulation Munnai Ilai Kudineer such as flavonoids, saponins or phenolic compounds may be responsible for the antinociceptive activities of this formulation. Since the findings of this study revealed a significant analgesic effect of the siddha formulation Munnai Ilai Kudineer, it can be concluded that terpenoids and specially saponins of siddha formulation Munnai Ilai Kudineermay be responsible for the observed analgesic effect which should be proved by further investigations.

It can be concluded that possesses anti-nociceptive properties which are probably mediated via inhibition of prostaglandin synthesis as well as central inhibitory mechanisms which may be of potential benefit for the management of pain and inflammatory disorders.

5.1.2 ANTI-INFLAMMATORY ACTIVITY

EVALUATION OF CARRAGEENAN INDUCED ANTI-INFLAMMATORY ACTIVITY OF SIDDHA FORMULATION MUNNAI ILAI KUDINEERIN ALBINO WISTAR RATS

Introduction

Inflammation is a complex biological response of vasculartissues against aggressive agents such as pathogens, irritants,or damaged cells. Acute inflammation is the initial responseand is characterized by the increased movement of plasmaand innate immune system cells, such as neutrophils andmacrophages, from the blood into the injured tissues. The stan-dard signs of inflammation are expressed by increased bloodflow, elevated cellular metabolism, vasodilatation, release ofsoluble mediators, extravasation of fluids and cellular influx. Upon the presence of the inflammatory agent, cell mem-branes induce the activation of phospholipase A2 followed byrelease of arachidonic acid and inflammatory mediators such ascytokines, serotonin,

histamine, prostaglandin and leukotrienesthat increase vascular permeability, thus facilitating the migra-tion of leukocytes to the site of inflammation. Inflammationinduced by carrageenan is acute, nonimmune, well-researched,and highly reproducible. Cardinal signs of inflammation—edema,hyperalgesia, and erythema—develop immediately following sub-E-mail address: sumana.sarkhel@yahoo.incutaneous injection, resulting from action of proinflammatoryagents—bradykinin, histamine, tachykinins, complement and reac-tive oxygen, and nitrogen species.Many saponins tested have displayed significant antinocicep-tive, anti-inflammatory and antipyretic activities possibly due totheir nonglycosidic moiety, the sapogenin, but also many diverseactivities have also been reported such as antiallergic, antifungal,analgesic and others . Moreover a variety of siddha formulation preparation haveproved to be useful in animal models of inflammation [7–10].

Paw swelling, or footpad edema, is a convenient method for assessing inflammatory responses to antigenic challenges and irritants. Typically, test compounds are assessed for acute anti-inflammatory activity by examining their ability to reduce or prevent the development of carrageenan-induced paw swelling. In the present study attempts are made to validate the claims of Munnai Ilai Kudineer regarding the anti-inflammatory activities of this siddha preparation.

Methods & materials

Animals

Male albino rats (180 ± 5 g) were obtained from animal house,K.M.College of pharmacy,maduarai and maintained in standard laboratory conditions. They were given standard labora-tory diet and water ad libitum. All animal experiments are approved by the Institutional Animal Ethics Committee, and were in accordance with the guidelines of the committee for the purpose of Control and Supervision of Experi-ments on Animal (CPCSEA), Government of India.

Acute inflammation

Carrageenan-induced rat paw oedema is used widely as a work-ing model of inflammation in the search for new anti-inflammatory drug. The anti inflammatory activity of the siddha formulation Munnai Ilai Kudineer was evaluated by carrageenan-induced rat paw oedema method [11]. Albino Wistar rats (180 ± 5 g) were used. Antiinflammatory activity was measured using carrageenan induced rat paw oedema assay. The rats were divided into 5 groups of 5 animals each. Group

I.were given normal saline and treated as negative control. Rats of Group II was treated with carragenan (1%w/v) in saline in the subplanter region of the right hind paw Rats in Group III were administered Indomethacin(10 mg/kg, bw) and considered as standard. Rats from Group IV and V were given two doses siddha formulation(100 and 200 mg/kg bw). Acute paw edema was induced by injecting 0.1 ml of 1% (w/v) carrageenan solution, pre-pared in normal saline. After 1 h, 0.1 ml, 1% carrageenan suspension in 0.9% NaCl solution was injected into the sub-plantar tissue of the right hind paw. The linear paw circumference will be measured at hourly interval for 4 h. The perimeter of paw was measured by using vernier callipers. Measurements were taken at 0–4 h after the administration of the carrageenan.

The anti-inflammatory activity was calculated by using the rela-tion

$$\%\text{inhibition of edema} = \frac{T - T0}{T} \times 100$$

T, Thickness of paw in control group; T0, Thickness of paw edema in the test compound treated group.

Carrageenan Induced Pleurisy In Rats

The animals were divided into five groups of five rats each as described in the carrageenan induced pawedema model [12,13] and each were pretreated with siddha formulation(100 and 200 mg/kg, p.o.), Indomethacin(10 mg/kg, p.o.) or normal saline (0.1 ml).One hour later all the animals were received 0.25 ml ofan intrapleural injection of 1 % carrageenan on the rightside of the thorax. The animals were sacrificed 3 h aftercarrageenan injection by ether inhalation. One ml ofheparinized Hank's solution was injected into the pleuralcavity and gently massaged to mix its contents. Thefluid was aspirated out of the cavity and the exudateswere collected. The number of migrating leukocytes inthe exudates was determined with Neubauer chamber.

The values of each experimental group were expressedas mean ± SEM and compared with the control group.

Statistical analysis

Results of antiinflammatory activity were expressed as Mean increase in paw diameter ± SD. Results were analyzed using one way ANOVA. Differences were considered as statistically significant at $P < 0.05$ are compared to control.

Table 5.1.2(a) Effect of siddha formulation Munnai Ilai Kudineer on Carrageenan Induced Rat Paw Edema.

Treatment	Dose (mg/kg, p.o.)	Mean increase in paw volume (ml)	% Decrease in paw volume
Normal control	10ml/kg saline	1.05 ± 0.09	
Toxic control	0.1 ml, 1% carrageenan	3.45 ± 0.24*a	
Standard control	10mg/kg Indomethacin	1.20 ± 0.12*b	65.21%
Treatment control	100mg/kg Munnai Ilai Kudineer	1.40 ± 0.18*b	59.42%
Treatment control	200mg/kg Munnai Ilai Kudineer	1.32 ± 0.14*b	61.73%

* (a) Values are significantly different from normal control G1 at P<0.01.

* (b) Values are significantly different from Toxic control G2 at P<0.01.

Table 5.1.2.b. Effect of siddha formulation Munnai Ilai Kudineeron Carrageenan Induced Pleurisy in Rats.

Treatment	Dose (mg/kg, p.o.)	Pleural exudates (ml)	Leukocytes (×103 cells/ml)
Normal control	10ml/kg saline	0.14±0.05	0.37±0.03
Toxic control	0.1 ml, 1% carrageenan	0.42±0.14*a	4.20±0.36*a
Standard control	10mg/kg Indomethacin	0.15±0.06*b	0.46±0.05*b
Treatment control	100mg/kg Munnai Ilai Kudineer	0.21±0.09*b	0.54±0.08*b
Treatment control	200mg/kg Munnai Ilai Kudineer	0.16±0.07*b	0.51±0.06*b

(a) Values are significantly different from normal control G1 at P<0.01

(b) Values are significantly different from Toxic control G2 at P<0.01.

Anti-inflammatory Activity of siddha formulation Munnai Ilai Kudineer

The above Table no 5.2 (a) showed the effect of siddha formulation Munnai Ilai Kudineeron carrageenan-induced edema in rats. Theresults obtained indicate that the siddha formulation Munnai Ilai Kudineer hadsignificant anti-inflammatory activity in rats. The siddha formulation Munnai Ilai Kudineer reduced the edema induced bycarrageenan by 59.42% and 61.73% on oral administration of 100 and 200 mg/kg, as compared to the untreated control group.

Indomethacinat 10 mg/kg inhibited the edema volume by 65.21%.

The aboveTable 5.2 (b) the effect of siddha formulation Munnai Ilai Kudineeron carrageenan-induced pleurisy in rats were (0.42±0.14 ml treated with the siddha formulation Munnai Ilai Kudineer(100 and 200 mg/kg, p.o.) decreased the pleuralexudates to 0.21±0.09 ml and 0.16±0.07.Treatment with Indomethacin(10 mg/kg, p.o.)produced the exudates of0.15±0.06 ml. The leukocyte count for the controlgroup was found to be $4.20\pm0.36\times10^3$ cells/ml. Animalstreated with the siddha formulation Munnai Ilai Kudineer and standard produced aleukocyte migration of $0.54\pm0.08\times10^3$,$0.51\pm0.06X10^3$ and $0.46\pm0.05\times10^3$ cells/ml, respectively.

DISCUSSION

Due to the increasing frequency of intake of NSAID's and their reported common side effects, thereis need to focus on the scientific exploration of siddha formulation drugs having fewer side effects. So, there is a continuoussearch for indigenous drugs, which can provide relief to inflammation. Carrageenan induced inflammation is a biphasicphenomenon. The first phase of edema is attributed to release of histamine and 5-hydroxytryptamine. Plateauphase is maintained by kinin like substances andsecond accelerating phase of swelling is attributed toprostaglandin like substances. The knowledge of thesemediators involved in different phases is important forinterpreting mode of drug action.The tests performed with the siddha formulation Munnai Ilai Kudineer in the pleurisy model showed that the siddha formulation Munnai Ilai Kudineer behaves as an inhibitor of leukocyte migration and theformation of pleural exudates when given orally, as reportedearlier.

Thus it can be concluded that the siddha formulation Munnai Ilai Kudineer possess significant anti-inflammatory activityin rats. Further studies involving the purification of the prerpation and the investigationsin the biochemical pathways may result in the developmentof a potent anti-inflammatory agent with a low (**Table 5.2.a&b**).

5.1.3.TOXICITY STUDY

Acute toxicity study of Munnai ilai kudineer

Cage Side Observations

Observations include changes in skin and fur, eyes and mucous membranes, and also respiratory, circulatory, autonomic and central nervous systems, and somatomotor activity and behavior pattern. Special attention is directed for the observation of tremors, convulsions, salivation, diarrhea, lethargy, sleep and coma.Body Weight, Food and Water Intake.Body weight, food and water intake are recorded at two-day intervals.

Pathology

Surviving animals are fasted overnight, weighed and humanely killed on the 15^{th} day using anesthetic ether. All test animals are subjected to gross necropsy.

Subchronic test for Munnai ilai kudineer

This experiment evaluates the toxicity potential ofMunnai ilai kudineer.

Method:

Male and female Wistar rats weighing 180 ± 10 g are used for the present study. The animals are divided into five groups of six animals each. The dose of the preparation is calculated based on the body weight of the animal. The animals in Group I are administered with a single daily dose of 0.5 ml of Tween 80 orally for 20 days. The animals in Group II are administered with 50 $mg.kg^{-1}$ b.w. of the Munnai ilai Kudineer orally once daily for 20 days. The animals in Group III are administered with 100 $mg.kg^{-1}$ b.w. of the Munnai ilai kudineerorally once daily for 20 days. The animals in Group IV and V are administered once daily with 200 and 400 $mg.kg^{-1}$ b.w. of the Munnai ilai Kudineer respectively for 20 days orally (Pieme,*et al* 2006, Joshi, *et al* 2007, Mythilypriya, *et al.,* 2007)The animals are then weighed every five days, from the start of the treatment, to record the weight variation. At the end of the treatment, blood samples are collected by puncturing retro orbital plexus after mild anesthesia for biochemical analysis. The collected blood sample is centrifuged within 5 min of collection at 4000 g for 10 min to obtain plasma, which is analyzed for total cholesterol, total triglyceride, HDL-cholesterol levels, LDL-cholesterol, plasma glucose, alanine aminotransferase (ALT), aspartate aminotransferase (AST), creatinine and urea.

Results

Acute toxicity study with Munnai ilai kudineer

The acute toxicity of Munnai ilai kudineerwas evaluated usingOECD- 423 guidelines. There was no mortality or morbidity observed in animals through the 15-days period following single oral administration at all selected dose levels of the Munnai ilai kudineer(Table-5.3.a). The animals did not show any changes in the general appearance during the observation period. Morphological characteristics such as fur, skin, eyes and nose appeared normal. No tremors, convulsion, salivation, diarrhea, lethargy or unusual behaviors such as self mutilation, walking backward and so forth were observed. Gait and posture, reactivity to handling or sensory stimuli, grip strength was also normal.

Table.5.1.3.a Acute toxicity studyof Munnai ilai kudineeron experimental mice.

	Dose ($mg.kg^{-1}$)	**Sign of Toxicity ($ST.NB^{-1}$)**	**Mortality ($D.S^{-1}$)**
Group I	0	0/3	0/3
Group II	300	0/3	0/3
Group III	2000	0/3	3/3

The acute toxicityof Munnai ilai kudineeron experimental mice was tested using OECD-423 guidelines, where ST- sign of toxicity; NB- normal behaviour; D-died; S- survive. Values are expressed as number of animals (n=3).

Effect of Munnai ilai kudineer in Subchronic Toxicity

Munnai ilai kudineerwere evaluated for subchronic toxicity.

Effect of Munnai ilai kudineeron body weight changes in rats

The effect of Munnai ilai Kudineer was observed for their effect on the body weight changes from the study it was observed that, there was significant increase ($p<0.05$) in body weight in all the animals observed.

Table5.1.3.bThe effects of **Munnai ilai kudineer** on body weight changes in rats.

Treatment	Day 1	Day 5	Day 10	Day 20
Control	186.15±6.8	188.45 ±6.20	193.15 ±6.35	197.7±6.58
Munnai ilai kudineer **50 mg.kg^{-1}**	193.30 ±6.4	194.30 ±6.30	195.25 ±6.70	199.30±6.72*
Munnai ilai kudineer **100 mg.kg^{-1}**	185.35 ±5.7	190.30 ±6.40	198.55 ±7.10	198.36±6.30*
Munnai ilai kudineer **200 mg.kg^{-1}**	194.30 ±7.2	199.15±6.50	195.90 ±7.20**	207.45±7.26**
Munnai ilai kudineer **400 mg.kg^{-1}**	190.65 ±6.05	193.15 ±5.60	192.60 ±6.35**	208.66±7.38**

A study on the effects of Munnai ilai kudineeron body weight changes in rats was carried out.. where, group I animals (GPI) were treated with normal saline (5 ml.kg^{-1}), group II animals (GPII) with 50 mg.kg^{-1} of Munnai ilai kudineer, group III animals (GPIII) with 100 mg.kg^{-1} of Munnai ilai kudineer, group IV animals (GPIV) with 200 mg.kg^{-1} of Munnai ilai kudineer,group V animals (GPV) with 400 mg.kg^{-1}Munnai ilai kudineer. According to **Table no 5.1.3 a&b** the values are expressed as mean ± S.E.M. n=6. The results of group I were compared with other groups such as II, III, IV, and V. The statistical analysis was carried out using one way ANOVA method, where **P<0.01 *P<0.05.

Effect of Munnai ilai kudineeron kidney,heart, liver and brain in rats

The table no 5.3c expressed the effects of **Munnai ilai kudineer**on kidney, heart, liver and brainof the rats were observed.From the study it was clear that, significant (p<0.01) changes in the weights of various organs of the animals occurred with higher doses of the extract (400 mg.kg^{-1} bwt), but macroscopic examinations did not show any changes in colour of the organs of the treated animals compared with the control.

Table.5.1.3.c The results are shown in Internal organs:

Treatment	Heart (gms)	Kidney (gms)	Liver (gms)	Brain (gms)
Control	0.37 ± 0.05	0.66± 0.03	3.31± 0.05	0.64± 0.05
Munnai ilai kudineer @50 mg.kg^{-1}	0.38± 0.02	0.82± 0.03	3.41± 0.03	0.67± 0.3
Munnai ilai kudineer @100 mg.kg^{-1}	0.39± 0.06	0.80± 0.04	3.33±0.02	0.65± 0.2
Munnai ilai kudineer @ 200 mg.kg^{-1}	0.38± 0.04	0.75± 0.02	3.31± 0.02	0.72± 0.05
Munnai ilai kudineer @400 mg.kg^{-1}	0.37± 0.03	0.76± 0.03	3.34± 0.03	0.76± 0.05

Table.5.1.3.cThe effects of **Munnai ilai kudineer** on kidney, heart, liver and brain of the rats. A study on the effects of **Munnai ilai kudineer** on kidney, heart, liver and brainof the rats was tested. where, group I animals (GPI) treated with normal saline (5 ml.kg^{-1}), group II animals (GPII) with 50 mg.kg^{-1} of **Munnai ilai kudineer**, group III animals (GPIII) with 100 mg.kg^{-1} of **Munnai ilai kudineer**, group IV animals (GPIV) with 200 mg.kg^{-1} of **Munnai ilai kudineer**, group V animals (GPV) with 400 mg.kg^{-1}**Munnai ilai kudineer**. The values are expressed as mean ± S.E.M. n=6. The results of group I were compared with other groups such as II, III, IV, and V. The statistical analysis was carried out using one way ANOVA method, where **P<0.01.

Effect of Munnai ilai kudineeron biochemical profiles of rats

The effect of Munnai ilai kudineer on various biochemical parameters of the experimental animal 'rats' were tested.From the study it was evident that, there was significant decrease ($p<0.05$) in the plasma glucose level in treated rats especially at higher dose (400 mg.kg^{-1}) compared with control rats. The control rats were administered only with 5 ml of normal saline. Significant decrease ($p<0.05$) in the plasma total cholesterol (TC), triglyceride (TG) and LDL-cholesterol levels were observed. But a significant increase ($p<0.05$) in HDL-cholesterol levels were observed in all the treated animals compared with the control animals. AST, ALT and ALP levels were also normal in the Munnai ilai kudineertreated animals. From the results of biochemical study there was no evidence of severe toxicity associated with the administration of higher concentration of Munnai ilai kudineer.

Table.5.1.3.e.The Biochemical results

Treatment	Glucose ($mg.dl^{-1}$)	Cholesterol ($mg.dl^{-1}$)	Triglyceride ($mg.dl^{-1}$)	HDL ($mg.dl^{-1}$)	LDL ($mg.dl^{-1}$)
Control	96.65± 0.62	39.62± 0.56	28.25± 0.45	138.25± 0.55	87.15±1.72
Munnai ilai kudineer@ 50 $mg.kg^{-1}$	94.50± 0.56	27.85± 0.25*	13.22± 0.23*	178.28± 0.65*	74.59±1.28
Munnai ilai kudineer@ 100 $mg.kg^{-1}$	91.45± 0.47	28.74± 0.26*	15.42± 0.28*	168.18±0.78*	71.84±1.10
Munnai ilai kudineer@ 200 $mg.kg^{-1}$	92.25± 0.55**	35.18± 0.30	17.84± 0.38*	187.30± 0.84*	50.60±1.30
Munnai ilai kudineer@ 400 $mg.kg^{-1}$	88.25± 0.45**	34.78± 0.28	19.28± 0.34*	185.2± 0.85*	49.50±0.84

According to Table.5.1.3.ethe effect of Munnai ilai kudineeron biochemical parameters such as glucose, cholesterol, triglyceride, HDL and LDL. A study on the effect of Munnai ilai kudineeron biochemical parameters such as glucose, cholesterol, triglyceride, HDL and LDL in rats was tested.where, group I animals (GPI) treated with normal saline (5 $ml.kg^{-1}$), group II animals (GPII) with 50 $mg.kg^{-1}$ of Munnai ilai kudineer, group III animals (GPIII) with 100 $mg.kg^{-1}$ of Munnai ilai kudineer, group IV animals (GPIV) with 200 $mg.kg^{-1}$ of, group V animals (GPV) with 400 $mg.kg^{-1}$Munnai ilai kudineer. The values are expressed as mean ± S.E.M. n=6. The results of group I were compared with other groups such as II, III, IV, and V. The statistical analysis was carried out using one way ANOVA method, where **P<0.01 *P<0.05

Table.5.1.3.f The effects of Munnai ilai kudineeron biochemical parameters such as AST, ALT, ALP, TP and Albumin in rats. A study on the effects of Munnai ilai kudineeron biochemical parameters.

Treatment	AST ($IU.l^{-1}$)	ALT ($IU.l^{-1}$)	ALP ($IU.l^{-1}$)	TP ($g.l^{-1}$)	ALBUMIN ($g.l^{-1}$)
Control	320.5±12.40	75.5± 3.18	256.58± 8.80	74.85± 3.32	39.15±2.35
Munnai ilai kudineer@50 mg.kg^{-1}	309.0±9.50**	73.5± 2.20**	269.10± 2.75**	74.30± 2.32	36.30±2.65
Munnai ilai kudineer@ 100 mg.kg^{-1}	310.3±7.20**	71.1± 3.15**	263.18± 6.70**	84.15± 2.82	38.30±3.05
Munnai ilai kudineer@ 200 mg.kg^{-1}	305.4±7.95	66.4± 2.90	268.00± 5.20	69.25± 3.32	40.20±2.75
Munnai ilai kudineer@ 400 mg.kg^{-1}	315.2± 8.20	68.3± 3.52	272.40± 4.40	76.05± 2.58	39.48±2.70

The effects of **Munnai ilai kudineer** on biochemical parameters such as AST, ALT, ALP, TP and Albumin in rats. A study on the effects of **Munnai ilai kudineer** on biochemical parameters such as AST, ALT, ALP, TP and Albumin rats was tested. where, group I animals (GPI) were treated with normal saline (5ml.kg^{-1}), group II animals (GPII) with 50 mg.kg^{-1} of HAEBD group III animals (GPIII) with 100 mg.kg^{-1} of **Munnai ilai kudineer**, group IV animals (GPIV) with 200 mg.kg^{-1} of **Munnai ilai kudineer**, and group V animals (GPV) with 400 mg.kg^{-1}**Munnai ilai kudineer** The values are expressed as mean ± S.E.M. n=6. The results of group I were compared with other groups such as II, III, IV, and V. The statistical analysis was carried out using one way ANOVA method, where **P<0.01 *P<0.05 (Table no5.1.3 f)

Effect of Munnai ilai kudineeron haematological parameters in rats

The effects of Munnai ilai kudineerwere observed for its effect on haematological parameterson the experimental rats. From the study it was evident that, a significant increase ($p<0.01$) were observed in the haemoglobin contents and RBC count in the group treated with 200 $mg.kg^{-1}$ body weight of Munnai ilai kudineerand a significant decrease of the parameters occurred in the group treated with 400 $mg.kg^{-1}$ b.w.t compared with the control. There was no significant change in the calcium level in all the treated animals compared to the control.

Table.5.1.3.g. The effect of **Munnai ilai kudineer** on haematological parameters such as HB, Calcium, RBC and WBC in rats.

Treatment	Haemoglobin ($mg.dl^{-1}$)	RBC (10^6 /mm^3)	WBC (10^6 /mm^3)	Calcium ($mg.dl^{-1}$)
Control	15.3± 0.25	9.15± 0.02	11.45± 0.05	9.45 ±0.02
Munnai ilai kudineer@ 50 $mg.kg^{-1}$	16.5± 0.26*	9.50± 0.04*	9.55± 0.01*	9.21 ±0.02
Munnai ilai kudineer@ 100 $mg.kg^{-1}$	16.3± 0.15*	9.55± 0.02*	8.354± 0.32*	9.27 ±0.20
Munnai ilai kudineer@ 200 $mg.kg^{-1}$	14.7± 0.20*	8.32± 0.12*	11.45± 0.03*	9.61 ±0.13
Munnai ilai kudineer@ 400 $mg.kg^{-1}$	13.05± 0.35*	8516± 0.45*	10.55± 0.13*	9.75 ±0.02

The above table no 5.1.3.g. The effect of Munnai ilai kudineer on haematological parameters such as HB, Calcium, RBC and WBC in rats. A study on the effect of Munnai ilai kudineeron haematological parameters such as Hb, RBC, WBC, Calcium in rats was tested. where, group I animals (GPI) treated with normal saline (5 $ml.kg^{1}$), group II animals (GPII) with 50 $mg.kg^{-1}$ of Munnai ilai kudineer, group III animals (GPIII) with 100 $mg.kg^{-1}$ of Munnai ilai kudineer, group IV animals (GPIV) with 200 $mg.kg^{-1}$ of Munnai ilai kudineer, and group V animals

(GPV) with 400 mg.kg^{-1}Munnai ilai kudineer. The values are expressed as mean ± S.E.M. n=6. The results of group I were compared with other groups such as II, III, IV and V. The statistical analysis was carried out using one way ANOVA method, where *P<0.05.

Discussion

The evaluation of sub-chronic and chronic dosing in experimental animals may be more relevant in determining the overall toxicity of the plant preparation. The highest overall concordance of toxicity in animals in comparison with humans is with hematological, gastrointestinal, and cardiovascular adverse effects whiles certain adverse effects in humans, especially hypersensitivity and idiosyncratic reactions, are poorly correlated with toxicity observed in animals (Table no 5.1.3.g).

In the present study, where the acute toxicity study of **Munnai ilai kudineer** was carried out as per OECD-423 guidelines,no mortality was observed in both the animals of control group as well as animals treated with a maximum dose of 2000 mg.kg^{-1}. Hence, 1/10th of 2000 mg.kg^{-1} i.e. 200 mg.kg^{-1} of dose was selected as a minimum dose for sub-acutetoxicity study (Abu Taha Nael *et al.* 2008)

The results of sub-acute toxicity study shows(Table no 5.1.3.g) that there was no significant change in animal behaviour due to the absence of toxicity. The animals treated with **Munnai ilai kudineer** showed normal growth pattern and body weight compared with control rats treated with normal saline. So the changes in body weight can be used as an indicator of adverse effects of drugs and chemicals (Tofovic and Jackson 1999; Raza*et al.* 2002; Teo 2002).

The changes in enzymes like ALP, AST and ALT levels show liver impairment, due to toxicity (Hayes, 1989). Serum cholesterol and proteins mainly regulated via synthesis in the liver and increase or decrease in serum concentrations of constituents suggest liver toxicity. The results of the present study were assessed after 28 days of administration of **Munnai ilai kudineer**, and it was found that **Munnai ilai kudineer**at all concentrations do not produce liver damage.

There was a slight decrease in plasma glucose level, when higher doses of **Munnai ilai kudineer**(400 mg.kg^{-1}) were administered in the treated rats..

Analysis of blood parameters is likely to risk evaluation as the change in hematological system has a higher predictive value for human toxicity, when data are translated from animal studies (Olson *et al*.2000). After 28 days of treatment, there were no significant changes in the haematological parameters between control and

treated groups. No significant changes in the levels of WBC, RBC were observed between control and test groups following repeated administration of **Munnai ilai kudineer**. Interestingly, significant increase in the levels of hemoglobin was found in treatment with **Munnai ilai kudineer** with a higher dose of 400 $mg.kg^{-1}$. The possible reason could be that one of the constituents **Munnai ilai kudineer** may increase absorption of iron.

The overall results suggest that **Munnai ilai kudineer** are non toxic to the haaematopoietic and leucopoietic system. The haematopoietic and leucopoietic systems are the most sensitive targets for toxic compounds and an important index of physiological and pathological status in man and animal (Adeneye*et al*. 2006). Therefore, it is possible to assume that the **Munnai ilai kudineer**is non haematotoxic

5.1.4 PHYTOCHEMICAL ANALYSIS

Analysis of the siddha preparation Munnai Ilai Kudineer

The siddha preparation Munnai Ilai Kudineer was prepared and used for phytochemical analysis.

Preliminary test, on the siddha preparation Munnai Ilai Kudineer was carried out for the presence of alkaloids, carbohydrates, glycosides, phytosterols, saponins, tannins, phenolic compounds, proteins and free amino acids, flavanoids, lignin, fixed oils and fats. The methods adopted for the estimation are as follows:

Test for Alkaloids (Evans, 1997)

A small segment of the siddha preparation Munnai Ilai Kudineer was mixed separately with a few drops of dilute hydrochloric acid and filtered. The filtrates were tested carefully with various alkaloidal reagents as follows:

a) *Mayer's test* (Evans, 1997): To a few ml of filtrate, a drop of Mayer's reagent is added by the side of the test tube. A white or creamy precipitate indicates that the test as positive.

b) *Hager's test* (Wagner *et al*., 1996): To a few ml of filtrate, one to 2ml of Hager's reagent is added. A prominent yellow precipitate indicates the test as positive.

c) *Dragendorff's test* (Waldi, 1965): To a few ml of filtrate, one to 2ml of Dragendorff's reagent is added. A prominent yellow precipitate indicates the test as positive.

Test for Carbohydrates (Ramakrishnan *et al*., 1994)

A small quantity of siddha preparation Munnai Ilai Kudineer was dissolved separately in 5ml of distilled water and filtered. The filtrate was subjected to Molisch's test to detect the presence of carbohydrates. Filtrate was treated with 2-3 drops of 1% alcoholic alpha naphthol solution and 2ml of concentrated sulphuric acid was added along the sides of the test tube. Appearance of brown ring at the junction of 2 layers shows the presence of carbohydrates. 37

Test for Glycosides

The siddha preparation Munnai Ilai Kudineer was hydrolyzed with hydrochloric acid for few h on a water bath and the hydrolysate was subjected to Legal's and Borntrager's test to detect the presence of different glycosides.

(a) ***Legal's Test***: To the hydrolysate, one ml of pyridine and few drops of sodium nitro prusside solution were added and then it was made alkaline with sodium hydroxide solution. Appearance of pink to red color shows the presence of glycosides and aglycones.

(b) ***Borntrager's Test***: Hydrolysate was treated with chloroform and the chloroform layer was separated. To this equal quantity of dilute ammonia solution was added. Ammoniacal layer acquires pink color, shows the presence of glycosides (Evans, 1997).

Test for Phytosterols (Finar, 1986)

(a) ***Liebermann Burchard Test*****:** Small amount of the siddha preparation Munnai Ilai Kudineer was dissolved with 3ml of acetic anhydride, a few drops of glacial acetic acid and followed by the addition of few drops of concentrated sulphuric acid. Appearance of bluish green color shows the presence of phytosterols.

(b) ***Salkowski Test*****:** Small quantities of the siddha preparation Munnai Ilai Kudineer were dissolved in chloroform separately. This chloroform solution was added with few drops of concentrated sulphuric acid. The appearance of bluish green color shows the presence of phytosterols.

Test for Saponins (Kokate, 1999)

Frothing Test: The siddha preparation Munnai Ilai Kudineer was diluted separately with 20ml of distilled water and it was agitated on a graduated cylinder for 15min. Absence of the foam formation shows the devoid of saponins.

Test for Phenolic Compounds and Tannins (Mace, 1963)

Small quantities of siddha preparation Munnai Ilai Kudineer was dissolved separately in water and tested for the presence of phenolic compound and tannins. In the process of testing and treating, the following observations were noted:

a) Dilute ferric chloride solution (5%) gives a dark green color.

b) 10% aqueous potassium dichromate solution gives yellowish brown precipitate.

c) 10% lead acetate solution gives a white precipitate.

Test for Proteins and Free Amino Acids (Fisher, 1968; Ruthmann, 1970)

Small quantities of siddha preparation Munnai Ilai Kudineer was dissolved in few ml of water and the following reaction were carried out

(a) *Millon's Test* :To 2ml of filtrate, few drops of Millon's reagent were added. A white precipitate indicates the presence of proteins (Rasch and Swift, 1960).

(b) *Ninhydrin Test*: To 2ml of filtrate 2 drops of ninhydrin solution was added. A characteristic purple color indicates the presence of amino acids (Yasma and Ichikawa, 1953).

(c) *Biuret Test*: An aliquot of 2ml of filtrate was treated with a drop of 2% copper sulphate solution. To this, 1ml of ethanol (95%) was added, followed by excess of potassium hydroxide pellets, Pink color in the ethanol layer indicates the presence of protein (Gahan, 1984).

Test for Flavanoids

(a) ***Shinoda's Test*:** Small quantity of siddha preparation Munnai Ilai Kudineer was treated with alcohol to that a piece of magnesium was added followed by an addition of concentrated hydrochloric acid drop wise and heated. Appearance of magenta color shows the presence of flavanoids (Harborne, 1984).

(b) ***Florescence Test*:** Small quantity of Munnai Ilai Kudineer was dissolved separately in alcohol and a drop of that extract was placed on Whatman filter paper and observed under UV light. Florescence indicates the presence of flavanoids.

Tests for Lignin

Small quantities of Munnai Ilai Kudineer was dissolved separately in few ml of alcoholic solution of hydrochloric acid and phloroglucinol gives red color, which shows lignin is present.

Tests for Fixed oils and Fats

(a) ***Spot Test*:** A small quantity of siddha preparation Munnai Ilai Kudineer was placed between 2 filter papers. Oil stains produced with any extract shows the presence of fats and fixed oils in the Munnai Ilai Kudineer (Harborne, 1984).

***(b)Saponification Test*:** A small quantity of siddha preparation Munnai Ilai Kudineer was treated with few drops of 0.5N alcoholic potassium hydroxide along with 2 to 3 drops of phenolphthalein. Later the mixture is refluxed for about 2h. Soap formation indicates the presence of fats and fixed oils in the Munnai Ilai Kudineer.

PHYTOCHEMICAL STUDY OF MUNNAI ILAI KUDINEER

The Munnai Ilai Kudineer was subjected to qualitative chemical investigation Details of the various tests performed for the presence of phytoconstituents is shown in Table 5.1.4 a.

Tests		**Munnai Ilai Kudineer**
Mayer's test	**Alkaloids**	Present
Dragendroff's test		Present
Hager's test		Present
Molisch test Legal's test	**Carbohydrates and glycosides**	Present
Borntrager's test for		Present
anthraquinones		Present
Liebermann-Burchard test	**Phytosterols**	Present
Salkowski test		Present
Shinoda test Magnesium turnings and hydrochloric acid (Presence of red color)	**Flavanoids**	Present
Fluorescence test		Present
Ferric chloride test	**Tannins**	Present
Biuret test		Present
Lead acetate test		Present
Millon's test	**Proteins**	Present
Biuret test		Present
Ninhydrin test		Present
Spot test	**Fixed oils and fats**	absent
Saponification test		absent
Phloroglucinol test	**Lignin**	Present
Forthing test	**Saponins**	absent

5.1.5ANTIMICROBIAL ACTIVITY PROCEDURE

Antibacterial Activity Procedure:

Dilution :1mg in 1ml (0.1g in 1ml)

Test Organism:

The test microorganisms used for antimicrobial analysis ***Microorganism name***were purchased from Microbial Type Culture Collection and Gene Bank (*MTCC*)Chandigarh.The bacterial strains were maintained on Nutrient Agar (NA) and fungi on Sabouraud Dextrose Agar (SDA).

Nutrient Broth Preparation

Pure culture from the plate were inoculated into Nutrient Agar plate and sub cultured at 37ºC for 24 h. Inoculum was prepared by aseptically adding the fresh culture into 2 ml of sterile 0.145 mol/L saline tube and the cell density was adjusted to 0.5 McFarland turbidity standard to yield a bacterial suspension of 1.5×108 cfu/ml. Standardized inoculum Used for Antimicrobial test.

Antimicrobial Test:

The medium was prepared by dissolving 38 g of Muller Hinton Agar Medium (Hi Media) in 1000 ml of distilled water. The dissolved medium was autoclaved at 15 Lbs pressure at 121^0C for 15 min (pH 7.3). The autoclaved medium was cooled, mixed well and poured petriplates (25 ml/plate) the plates were swabbed with Pathogenic Bacteria culture viz.***Microorganism name*** Finally, The Sample or Sample loaded Disc was then placed on the surface of Mullar-Hinton medium and the plates were kept for incubation at 37°C for 24 hours. At the end of incubation, inhibition zones were examined around the disc and measured with transparent ruler in millimeters. The size of the zone of inhibition (including disc) was measured in millimeters. The absence of zone inhibition was interpreted as the absence of activity (Kohner *et al*., 1994; Mathabe *et al*., 2006). The activities are expressed as resistant, if the zone of inhibition was less than 7 mm, intermediate (8-10 mm) and sensitive if more than 11 mm (Assam *et al*., 2010)

ANTIFUNGi ASSAY BY DISC DIFFUSION METHOD (Bauer *et al*., 1966)

Antibiotic susceptibility tests were determined by agar disc diffusion (Kirby–Bauer) method. Fungi strains *(Fungi Name)* were swabbed using sterile cotton swabs in SDA agar plate. Up to 40 µl of each concentration of the extract were respectively introduced in the sterile discs using sterile pipettes. The disc was then placed on the surface of SDA medium and the compound was allowed to diffuse for 5 minutes and

the plates were kept for incubation at 22°C for 48 hours. At the end of incubation, inhibition zones were examined around the disc and measured with transparent ruler in millimeters.

Minimum Inhibitory Concentration (MIC) Determination

This assay consists in the determination of chemical agent spectrum of action, according to resistance of studied microorganisms. It was developed the determination of minimum inhibitory concentration (MIC) for every chemical agent, through the classic method of successive dilution. In twelve numbered screw tubes (10 x 100 mm), 1 mL of TSB (trypticase soy broth) medium was distributed for every tube, except for the tube number 1. The tubes were submitted to autoclave under constant pressure and temperature of 121 °C. For the first and the second tubes of the series, 1 mL of tested sanitizing agent was added; tube 2 was stirred and 1 mL was withdrawn and transferred for tube 3. This successive transference was repeated until tube 11. It was added to all flasks, except for flask number.

Figuire-1

Figure-2

Figure-3

Figure-4

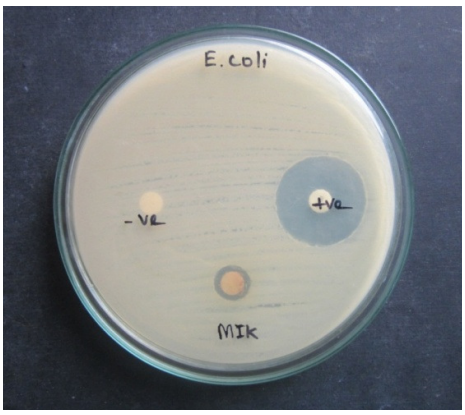


Figure-5

inbiotics
workshop for biologists

(A training unit affiliated to Institute of Biology and Clinical Research)

Name : **Dr. N.Prakash**

Sample received date : **11.06.2019**

Result Sending date : **24.06.2019**

ANTIMICROBIAL RESULTS

Sample Code	Bacteria Strains Name				
	Staphylococcus aureus (G+)	*Streptococcus mutans* (G+)	*Bacillus subtilis*(G+)	*Klebsilla pneumonia* (G-)	E – coli (G-)
MIK	14	13	11	12	10
PC	25	15	25	25	23
NC	-	-	-	-	-

Keys

PC	-	*Positive Control (Streptomycin)*
NC	-	*Negative Control*
-	-	*No Zone*
Mm	-	*Millimetre*
G+	-	*Gram Positive Organism*
G-	-	*Gram Negative Organism*

Dr.Johny Manoji

SCIENTIFIC OFFICER *INBIOTICS*

5.1.6 BIO-CHEMICAL ANALYSIS OF MUNNAI ILAI KUDINEER

Preparation of the extract:

5gms of the drug is weighed accurately and placed in a 250ml clean beaker then 50ml of distilled water is added and dissolved well. Then it is boiled well for about 10 minutes. It is cooled and filtered in a 100ml volumetric flask and then it is made to 100ml with distilled water. This extract is taken for analysis.

QUALITATIVE ANALYSIS

Sl. No.	Experiment	Observation	Inference
1.	**TEST FOR CALCIUM:** 2ml of the above prepared extract is taken in a clean test tube. To this add 2ml of 4% Ammonium oxalate solution.	No white precipitate is formed	Absence of Calcium
2.	**TEST FOR SULPHATE:** 2ml of the extract is added to 5% Barium chloride solution.	A white precipitate is formed	Indicates the presence of sulphate
3.	**TEST FOR CHLORIDE:** The extract is treated with silver nitrate solution.	A white precipitate is formed	Indicates the presence of chloride
4.	**TEST FOR CARBONATE:** The substance is treated with concentrated Hcl.	No brisk effervessence is formed	Absence of carbonate
5.	**TEST FOR STARCH:** The extract is added with weak iodine solution.	No Blue colour is formed	Absence of starch
6.	**TEST FOR FERRIC IRON:** The extract is acidified with Glacial acetic acid and potassium ferro cyanide.	No blue colour is formed	Absence of ferric iron
7.	**TEST FOR FERROUS IRON:** The extract is treated with concentrated nitric acid and ammonium thiocyanate solution.	Bloodred colour is formed	Indicates the presence of ferrous iron

8.	**TEST FOR PHOSPHATE:** The extract is treated with ammonium molybdate and concentrated nitric acid.	No yellow precipitate is formed	Absence of phosphate
9.	**TEST FOR ALBUMIN:** The extract is treated with Esbach's reagent.	No yellow precipitate is formed	Absence of albumin
10.	**TEST FOR TANNIC ACID:** The extract is treated with ferric chloride.	No blue black precipitate is formed	Absence of tannic acid
11.	**TEST FOR UNSATURATION:** Potassium permanganate solution is added to the extract.	It gets decolourised	Indicates the presence of unsaturated compound
12.	**TEST FOR THE REDUCING SUGAR:** 4ml of Benedict's qualitative solution is taken in a test tube and allowed to boil for 2 minutes and add 8-10 drops of the extract and again boil it for 2 minutes.	No colour change occurs	Absence of reducing sugar
13.	**TEST FOR AMINO ACID:** One or two drops of the extract is placed in a filter paper and dried well. After drying 1% ninhydrin is sprayed over the same and dried it well.	Violet colour is formed	Indicates the presence of Amino acid
14.	**TEST FOR ZINC:** The extract is treated with potassium ferrocyanide.	No white precipitate is formed	Absence of zinc

Inference:

Indicates the presence of sulphate, chloride, ferrous iron, unsaturated compound and amino acid.

5.2 CLINICAL STUDY

The results were observed regarding the following criteria by clinical trial study on 40 patients. 20 Out patients and 20 In patients in both sex were studied. Theselection criteriaare,

2. Age Distribution
3. Sex Distribution
4. Kaalam
5. Paruva kaalam
6. Thinai
7. Constitution of body
8. Gunam
9. Religion
10. Socio-Economical Status
11. Food Habits
12. Family History
13. Occupation
14. Aetiological Factors
15. Mode of Onset
16. Duration of Illness
17. Clinical Manifestation
18. Kanmenthiriyam
19. Gnanendrium
20. Kosam
21. Condition of Mukkutram

    a).Vatham b). Pitham c). Kapam
22. Involvement of Udal Thathukkal
23. Conditions of Envagai Thervugal
24. Neer Kuri
25. NeiKuri
26. Radiological Findings
27. Back Pain Functional Score Scale
28. Gradation of results

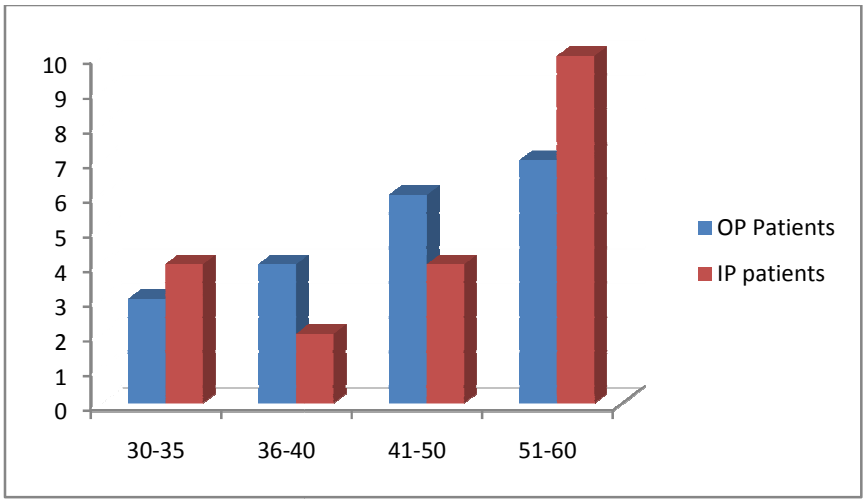
1. AGE DISTRIBUTION

Table-1 illustrates the age distribution of age and its percentage.

Sl. No.	Age group (In years)	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	30-35	3	15%	4	20%
2.	36-40	4	20%	2	10%
3.	41-50	6	30%	4	20%
4.	51-60	7	35%	10	50%
	Total	**20**	**100%**	**20**	**100%**

FIGURE-1

AGE DISTRIBUTION



From the above table-1 it is observed that the highest incidence of Thandaga Vatham in 20 Out patient is among the age group of 51-60 with 35% and among 20 In patients also in the age group of 51-60 with 50%.

2. SEX DISTRIBUTION

Table-2 illustrates the distribution of sex and its percentage.

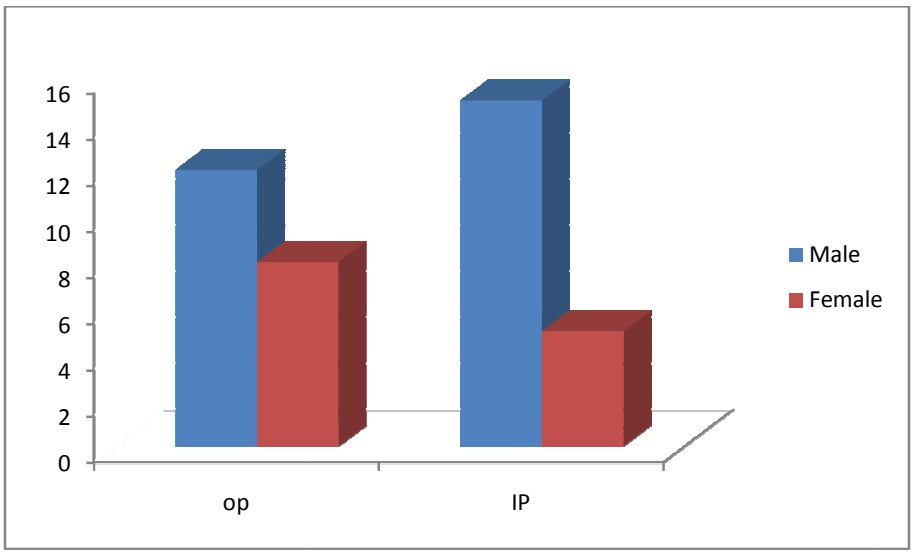
TABLE-2

SEX DISTRIBUTION

Sl. No.	Sex	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Male	12	60%	15	75%
2.	Female	8	40%	5	25%
	Total	**20**	**100%**	**20**	**100%**

FIGURE-2

SEX DISTRIBUTION



From the above table-2 it is observed that among 20 Out patients 60% were males and 40% were females and among 20 In patients 75% were males and 25% were females.

3. KAALAM

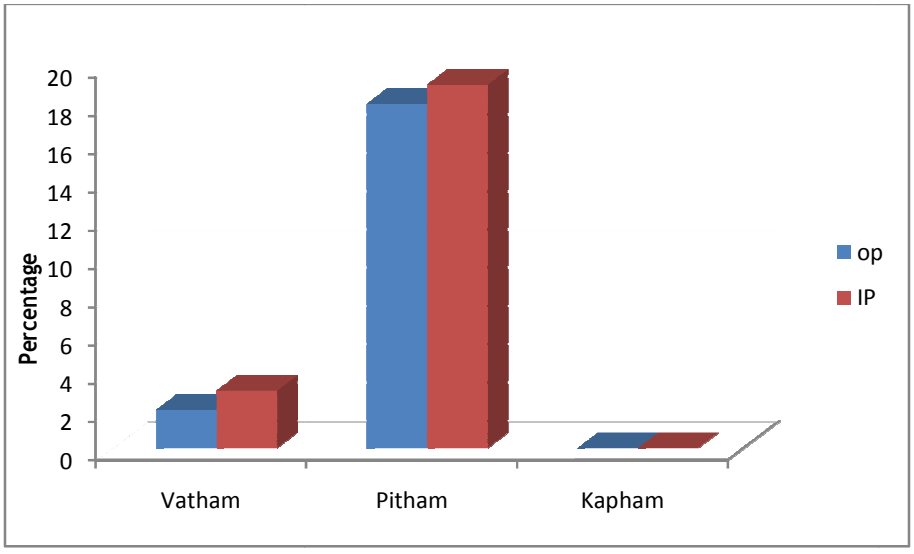
Table-3 illustrates the distribution of kaalam and its percentage.

TABLE-3 DISTRIBUTION OF KAALAM

Sl. No.	Kaalam	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Vatham	2	10%	3	15%
2.	Pitham	18	90%	17	85%
3.	Kapam	-	-	-	-
	Total	**20**	**100%**	**20**	**100%**

FIGURE-3

DISTRIBUTION OF KAALAM



From the above table-3 it is observed that the highest incidence of Thandaga Vatham among 20 Out patients is in Pitha Kaalam with 90% and among 20 In patients, is also Pitha Kaalam with 85%.

4. PARUVA KAALAM

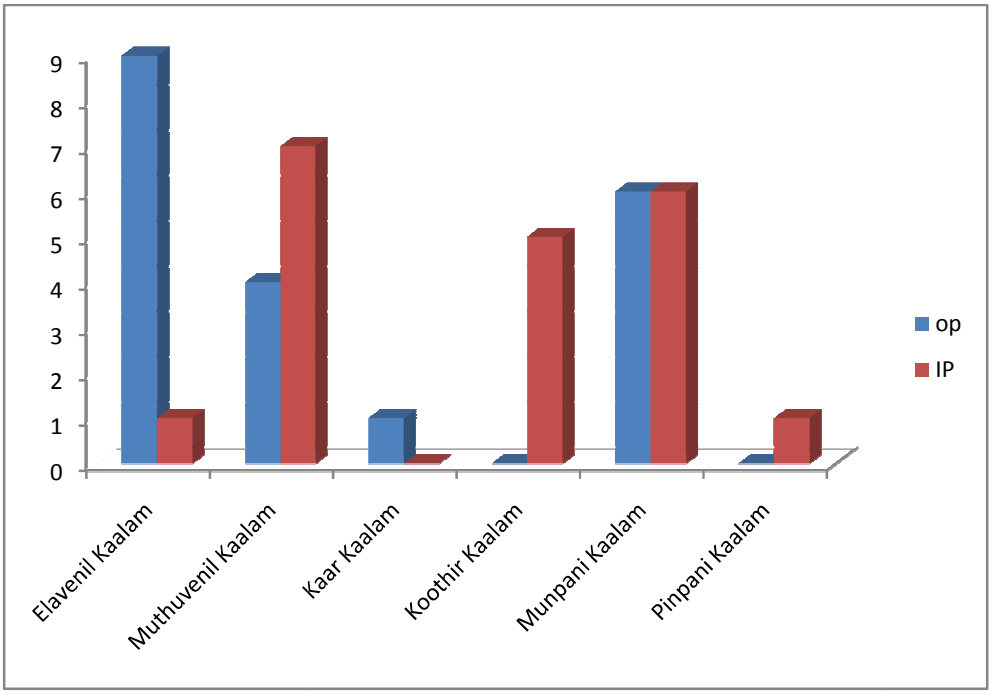
Table-4 illustrates the distribution of paruva kaalam and its percentage.

TABLE-4 DISTRIBUTION OF PARUVA KAALAM

Sl. No.	Paruva Kaalam	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Elavenil Kaalam	9	45%	1	5%
2.	Muthuvenil Kaalam	4	20%	7	35%
3.	Kaar Kaalam	1	5%	-	-
4.	Koothir Kaalam	-	-	5	25%
5.	Munpani Kaalam	6	30%	6	30%
6.	Pinpani Kaalam	-	-	1	5%
	Total	**20**	**100%**	**20**	**100%**

FIGURE-4

DISTRIBUTION OF PARUVA KAALAM



From the above table-4 it is observed that the highest incidence of Thandaga Vatham among 20 Out patients is in Elavenil Kaalam and among 20 In patient is in Muthuvenil Kaalam.

5. THINAI

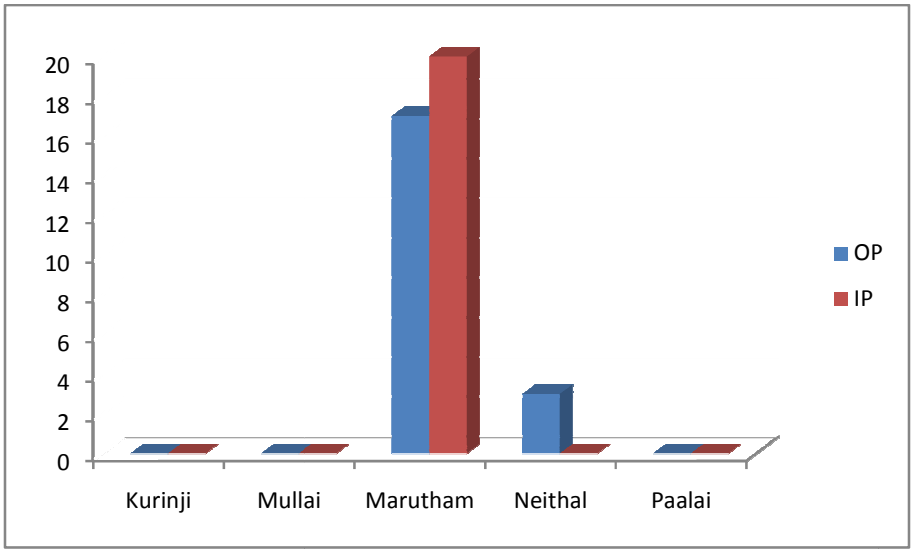
Table-5 illustrates the distribution of thinai and its percentage.

TABLE-5 DISTRIBUTION OF THINAI

Sl. No.	Thinai	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Kurinji	-	-	-	-
2.	Mullai	-	-	-	-
3.	Marutham	17	85%	20	100%
4.	Neithal	3	15%	-	-
5.	Paalai	-	-	-	-
	Total	**20**	**100%**	**20**	**100%**

FIGURE-5

DISTRIBUTION OF THINAI



From the above table-5 it is observed that highest incidence of Thandaga Vatham among 20 Out patients were in the Marutham Thinai with 85% and among 20 In Patients also in Marutham Thinai with 100%.

6. CONSTITUTION OF BODY

Table-6 illustrates the distribution of constitution of the body and its percentage.

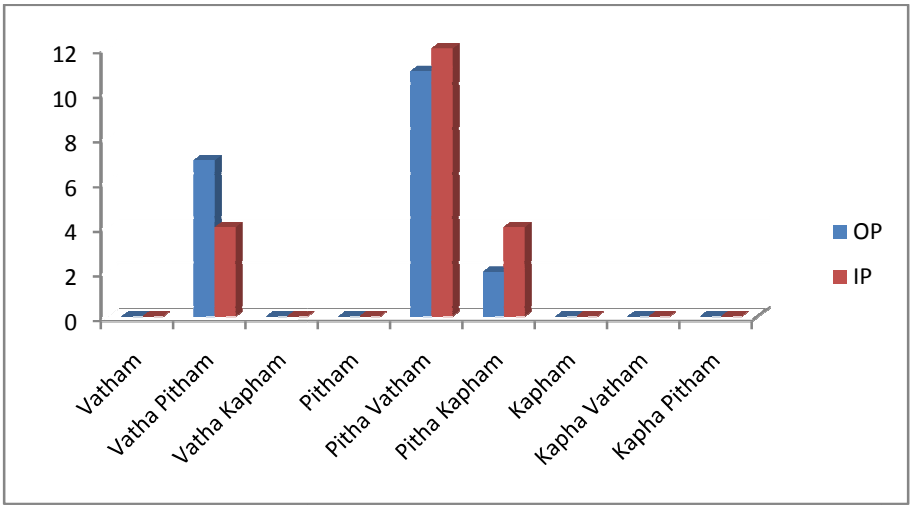
TABLE-6

DISTRIBUTION OF CONSTITUTION OF BODY

Sl. No.	Constitution of body	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Vatham	-	-	-	-
2.	Vatha Pitham	7	35%	4	20%
3.	Vatha Kapam	-	-	-	-
4.	Pitham	-	-	-	-
5.	Pitha Vatham	11	55%	12	60%
6.	Pitha Kapam	2	10%	4	20%
7.	Kapam	-	-	-	-
8.	Kaba Vatham	-	-	-	-
9.	Kaba Pitham	-	-	-	-
	Total	**20**	**100%**	**20**	**100%**

FIGURE-6

DISTRIBUTION OF CONSTITUTION OF BODY



From the above table-6 it is observed that the highest incidence of Thandaga Vatham among 20 Out patients is Pitha Vatham Thegi with 55% and among 20 In patients is also Pitha Vatham Thegi with 60%.

7. GUNAM

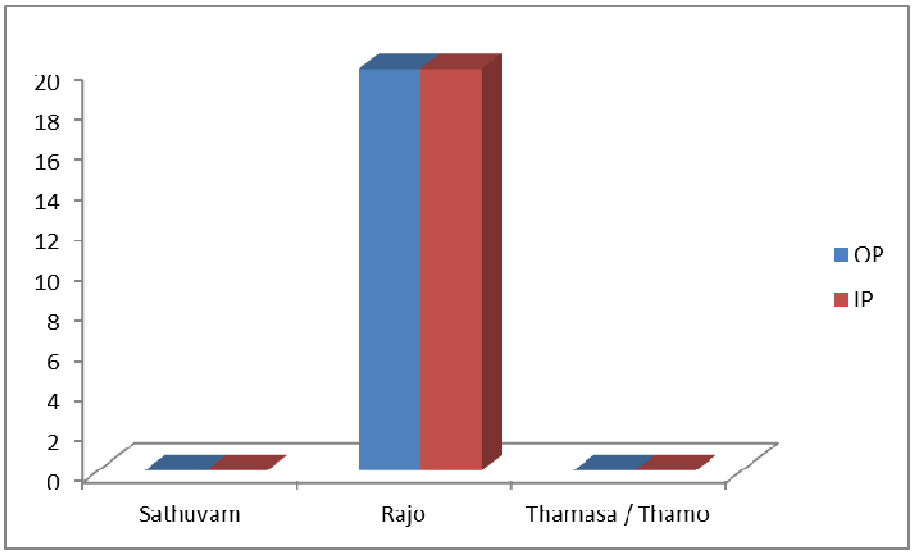
Table-7 illustrates the distribution of gunam and its percentage.

TABLE-7 DISTRIBUTION OF GUNAM

Sl. No.	Gunam	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Sathuvam	-	-	-	-
2.	Rajo	20	100%	20	100%
3.	Thamasa / Thamo	-	-	-	-
	Total	**20**	**100%**	**20**	**100%**

FIGURE-7

DISTRIBUTION OF GUNAM



From the above table-7 it is observed that the highest incidence of Thandaga Vatham among 20 Out patients and 20 In patients with cent percent belongs to Rajo Gunam.

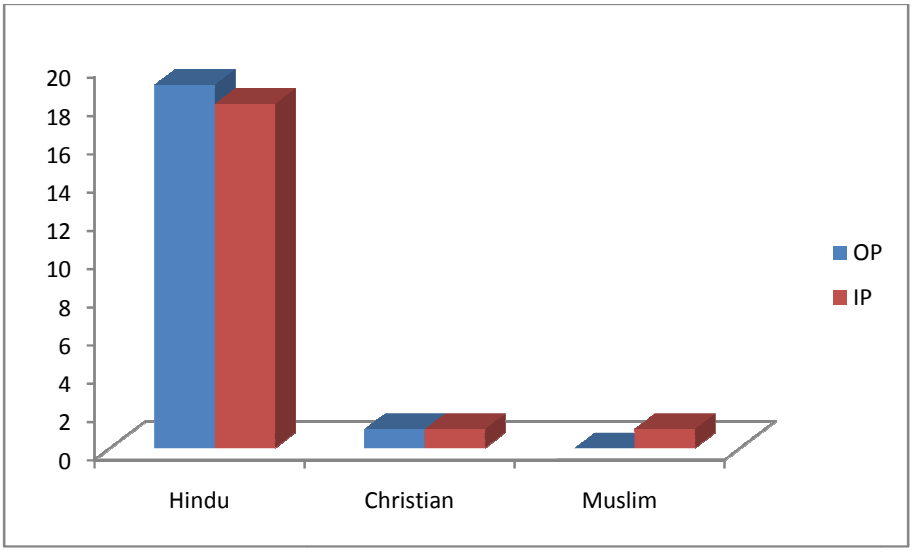
8. RELIGION

Table-8 illustrates the distribution of religion and its percentage.

TABLE-8 DISTRIBUTION OFRELIGION

Sl. No.	Religion	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Hindu	19	95%	18	90%
2.	Christian	1	5%	1	5%
3.	Muslim	-	-	1	5%
	Total	**20**	**100%**	**20**	**100%**

FIGURE-8 DISTRIBUTION OFRELIGION



From the above table-8 it is observed that among 20 Out patients 95% were Hindus, 5% wereChristians and among 20 In patients 90% were Hindus, 5% were Christians and 5% were Muslims.

9. SOCIO-ECONOMICAL STATUS

Table-9 illustrates the distribution of socio-economical status and its percentage.

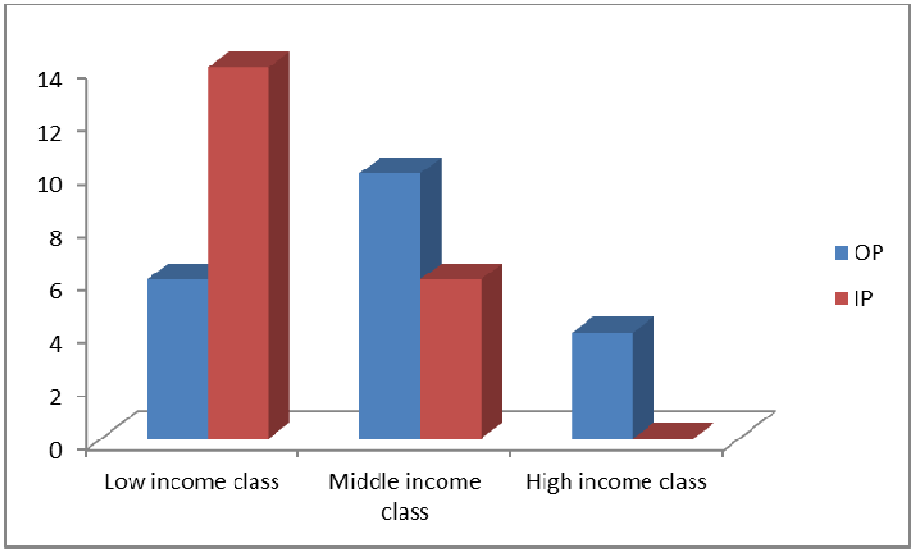
TABLE-9

DISTRIBUTION OF SOCIO-ECONOMICAL STATUS

Sl. No.	Socio-Economical Status	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Low income class	6	30%	14	70%
2.	Middle income class	10	50%	6	30%
3.	High income class	4	20%	-	-
	Total	**20**	**100%**	**20**	**100%**

FIGURE-9

DISTRIBUTION OF SOCIO-ECONOMICAL STATUS



From the above table-9 it is observed that the highest incidence of Thandaga Vatham among 20 Out patients is in Middle income class with 50% and among 20 In patients is in Low Income class with 70%.

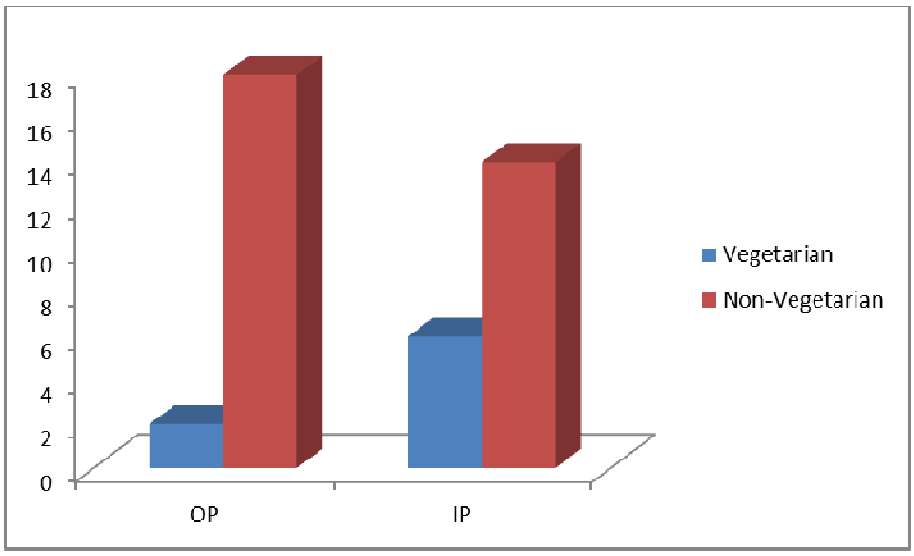
10. FOOD HABITS

Table-10 illustrates the distribution of diet and its percentage.

TABLE-10 DISTRIBUTION OF FOOD HABITS

Sl. No.	Food Habits	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Vegetarian	2	10%	6	30%
2.	Non-Vegetarian	18	90%	14	70%
	Total	**20**	**100%**	**20**	**100%**

FIGURE-10 DISTRIBUTION OF FOOD HABITS



From the above table-10 it is observed that the highest incidence of Thandaga Vatham among 20 Out patients is in Non-Vegetarians with 90% and among 20 In patients is also in Non-Vegetarians with 70%.

11. FAMILY HISTORY

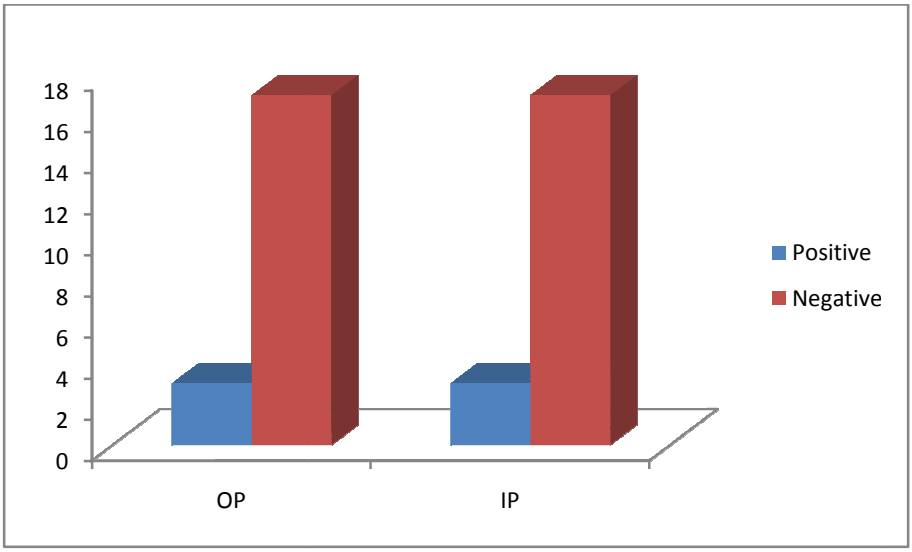
Table-11 illustrates the family history and its percentage.

TABLE-11 FAMILY HISTORY

Sl. No.	Family History	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Positive	3	15%	3	15%
2.	Negative	17	85%	17	85%
	Total	**20**	**100%**	**20**	**100%**

FIGURE-11

FAMILY HISTORY



From the above table-11 it is observed that among 20 Out patients and 20 In patients, 15% have positive family history and 85% don't have positive family history.

12. OCCUPATION

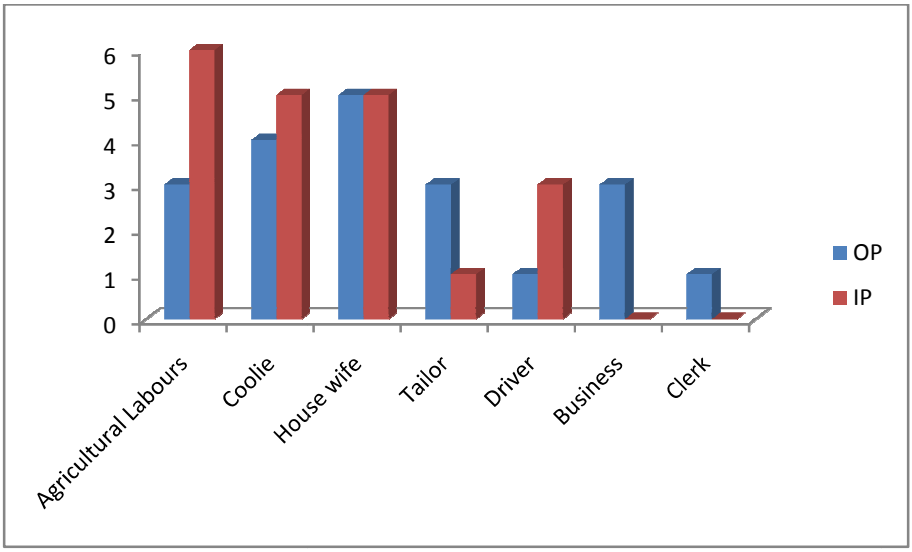
Table-12 illustrates the occupation and its percentage.

TABLE-12 OCCUPATION

Sl. No.	Occupation	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Agricultural Labours	3	15%	6	30%
2.	Coolie	4	20%	5	25%
3.	House wife	5	25%	5	25%
4.	Tailor	3	15%	1	5%
5.	Driver	1	5%	3	15%
6.	Business	3	15%	-	-
7.	Clerk	1	5%	-	-
	Total	**20**	**100%**	**20**	**100%**

FIGURE-12

DISTRIBUTION OF OCCUPATION



From the above table-12 it is observed that the highest incidence of Thandaga Vatham among 20 Out patients were house wives with 25% and among 20 In patients is also Agricultural Labours with 30%.

13. AETIOLOGICAL FACTORS

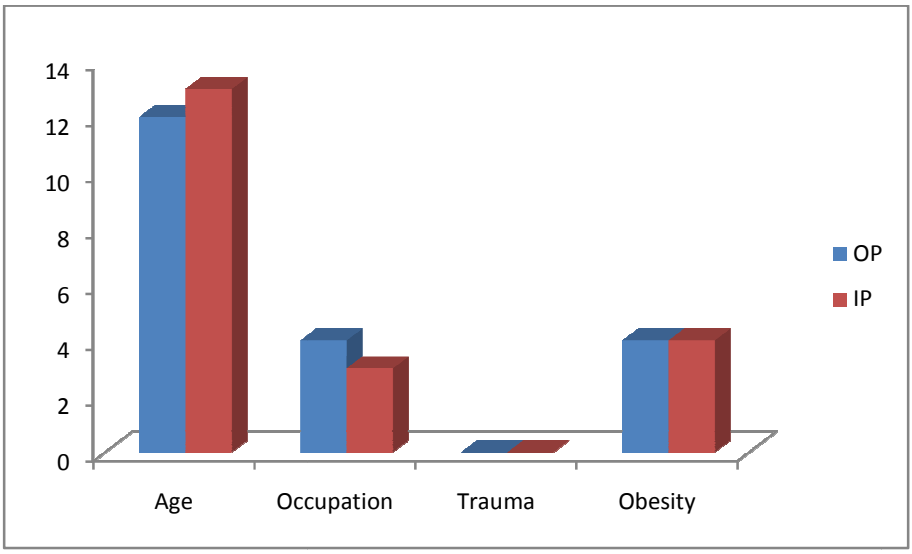
Table-13 illustrates the aetiological factors and its percentage.

TABLE-13 AETIOLOGICAL FACTORS

Sl. No.	Aetiological Factors	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Age	12	60%	13	65%
2.	Occupation	4	20%	3	15%
3.	Trauma	-	-	-	-
4.	Obesity	4	20%	4	20%
	Total	**20**	**100%**	**20**	**100%**

FIGURE-13

AETIOLOGICAL FACTORS



From the above table-13 it is observed that the highest incidence of Thandaga Vatham among 20 Out patients is due to age related aetiological factors with 60% and among 20 In patients is also due to age related aetiological factors with 65%.

14. MODE OF ONSET

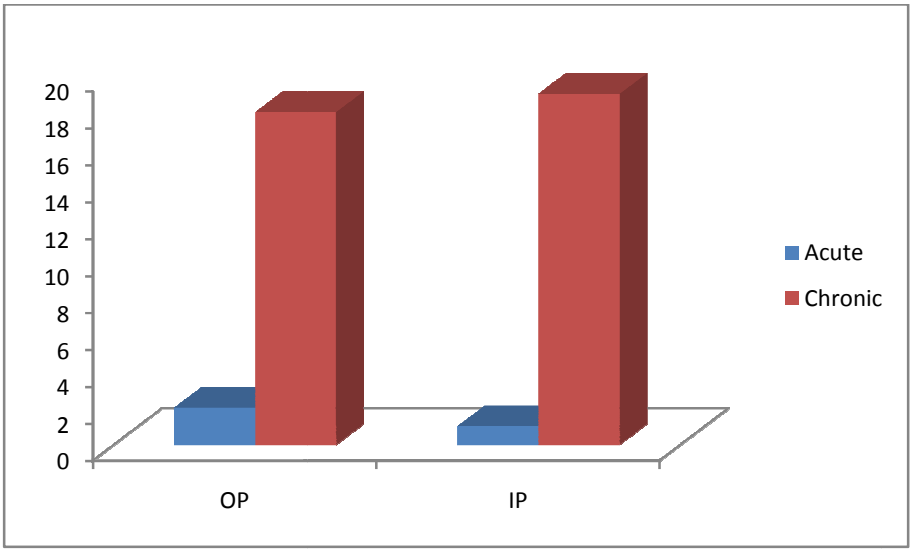
Table-14 illustrates the mode of onset and its percentage.

TABLE-14 MODE OF ONSET

Sl. No.	Mode of Onset	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Acute	2	10%	1	5%
2.	Chronic	18	90%	19	95%
	Total	**20**	**100%**	**20**	**100%**

FIGURE-14

MODE OF ONSET



From the above table-14 it is observed that among 20 Out patients, 90% were in chronic state and among 20 In patients, 95% were in chronic state.

15. DURATION OF ILLNESS

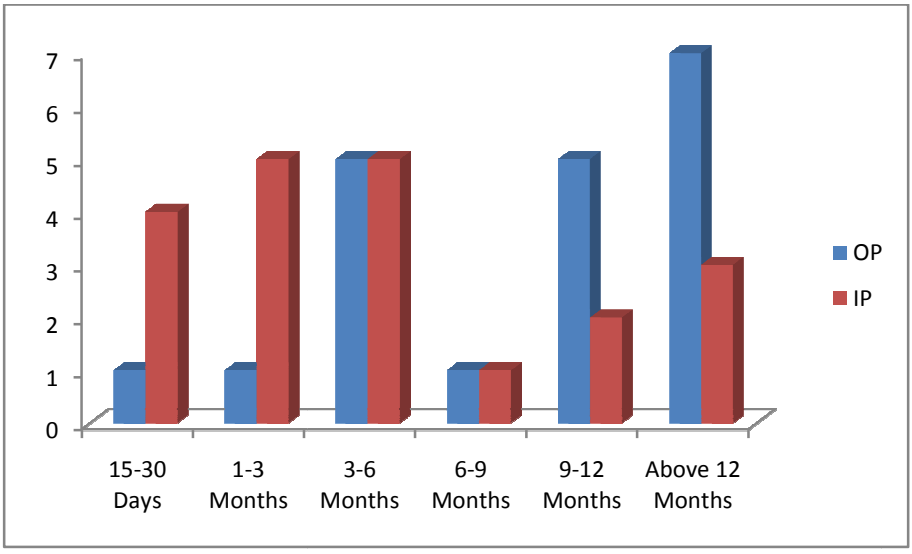
Table-15 illustrates the duration of illness and its percentage.

TABLE-15 DURATION OF ILLNESS

Sl. No.	Duration of illness	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	15-30 Days	1	5%	4	20%
2.	1-3 Months	1	5%	5	25%
3.	3-6 Months	5	25%	5	25%
4.	6-9 Months	1	5%	1	5%
5.	9-12 Months	5	25%	2	10
6.	Above 12 Months	7	35%	3	15%
	Total	**20**	**100%**	**20**	**100%**

FIGURE-15

DURATION OF ILLNESS



From the above table-15 it is observed that the highest duration of illness among 20 Out patientsAbove 12 Months and 20 In patients is 1-6 Months.

16. CLINICAL MANIFESTATION

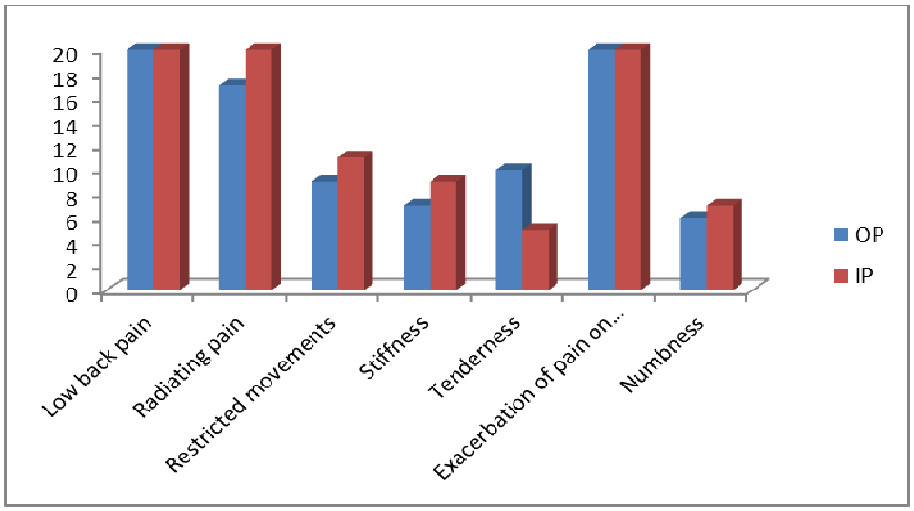
Table-16 illustrates the clinical manifestation and its percentage.

TABLE-16 CLINICAL MANIFESTATION

Sl. No.	Clinical Manifestation	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Low back pain	20	100%	20	100%
2.	Radiating pain	17	85%	20	100%
3.	Restricted movements	9	45%	11	55%
4.	Stiffness	7	35%	9	45%
5.	Tenderness	10	50%	5	25%
6.	Exacerbation of pain on movements	20	100%	20	100%
7.	Numbness	6	30%	7	35%

FIGURE-16

CLINICAL MANIFESTATION



From the above table-16 it is observed that, among 20 Out patients, 100% of cases have low back pain and exacerbation of pain on movements. 85% have radiating pain, 45% have restricted movements, 35% have stiffness, 35% have tenderness and 50% have numbness. Among 20 Inpatients 100% of cases have low back pain, radiating pain and exacerbation of pain on movements. 55% have restricted

movements, 45% have stiffness, 25% have tenderness and 35% have numbness.

17. KANMENTHIRIYAM

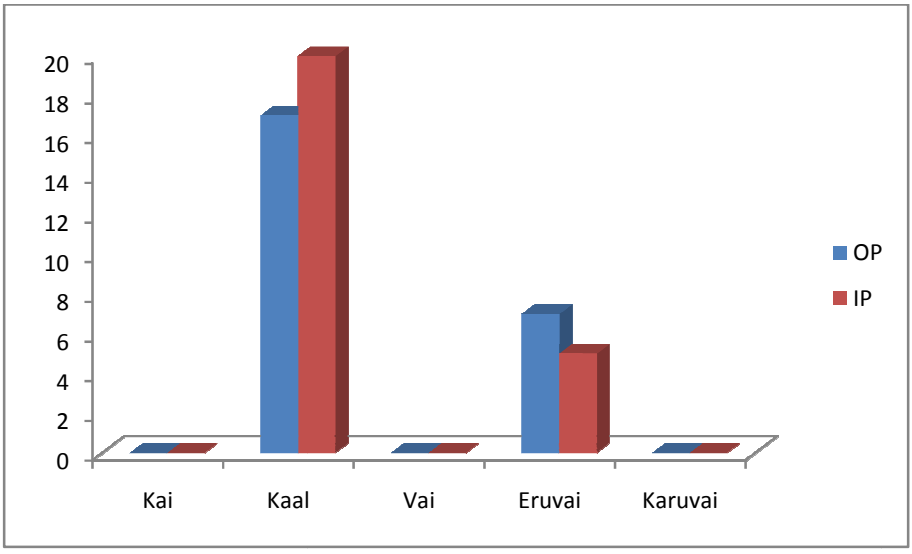
Table-17 illustrates the kanmenthiriyam and its percentage.

TABLE-17 KANMENTHIRIYAM

Sl. No.	Kanmenthiriyam	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Kai	-	-	-	-
2.	Kaal	17	85%	20	100%
3.	Vai	-	-	-	-
4.	Eruvai	7	35%	5	25%
5.	Karuvai	-	-	-	-

FIGURE-17

KANMENTHIRIYAM



From the above table-17 it is observed that among 20 Out patients, 85% were affected in Kaal and 35% in Eruvai. Among 20 In patients 100% were affected in Kaal and 25% in Eruvai.

18. GNANENDRIUM

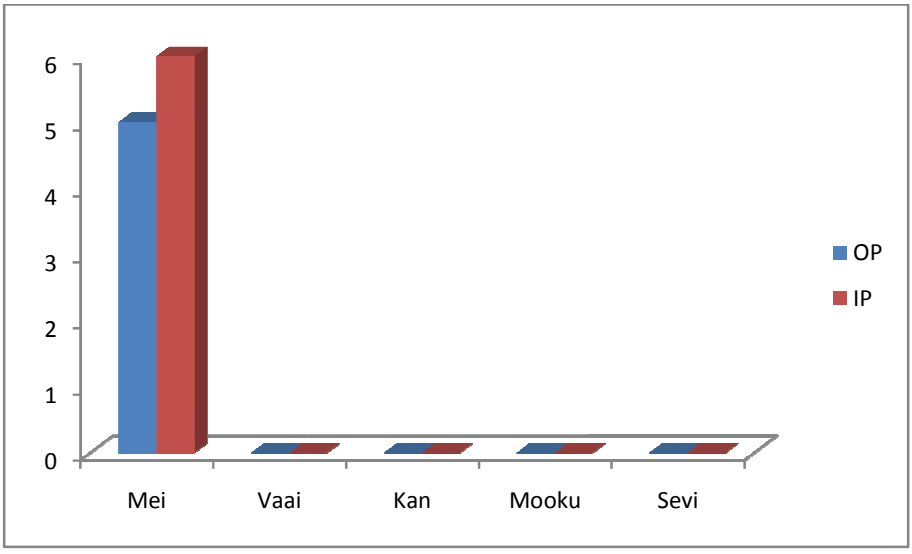
Table-18 illustrates the gnanendrium and its percentage.

TABLE-18 GNANENDRIUM

Sl. No.	Gnanendrium	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Mei	5	25%	6	30%
2.	Vaai	-	-	-	-
3.	Kan	-	-	-	-
4.	Mooku	-	-	-	-
5.	Sevi	-	-	-	-

FIGURE-18

GNANENDRIUM



From the above table-18 it is observed that, among 20 Out patients, 25% were affected in Mei, Among 20 In patients 30% were affected in Mei.

19. KOSAM

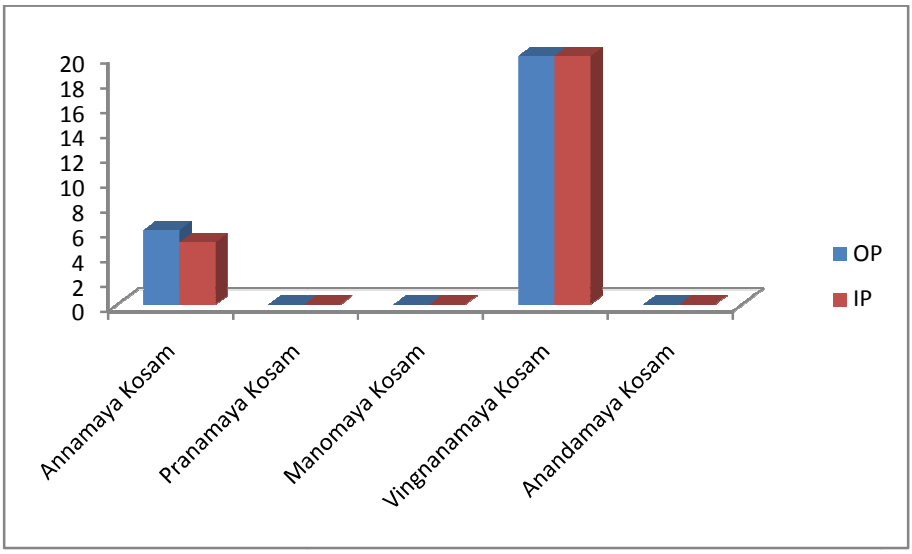
Table-19 illustrates the kosam and its percentage.

TABLE-19 KOSAM

Sl. No.	KOSAM	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Annamaya Kosam	6	30%	5	25%
2.	Pranamaya Kosam	-	-	-	-
3.	Manomaya Kosam	-	-	-	-
4.	Vingnanamaya Kosam	20	100%	20	100%
5.	Anandamaya Kosam	-	-	-	-

FIGURE-19

KOSAM



From the above table-19 it is observed that among 20 Out patients 100% were affected with Vingnanamaya Kosam and 30% were affected with Annamaya Kosam. Among 20 In patients, 100% were affected with Vingnanamaya Kosam and 25% were affected with Annamaya Kosam.

20 (a).CONDITION OF MUKKUTRAM (a). VATHAM:

Table-20 (a) illustrates the condition of vatham and its percentage.

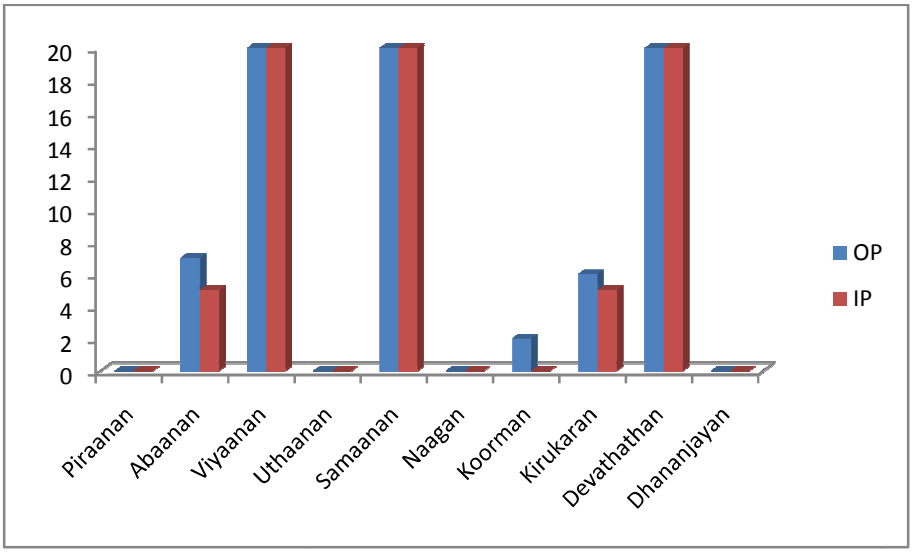
TABLE-20 (a)

CONDITION OF VATHAM

Sl. No.	Condition of Vatham	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Piraanan	-	-	-	-
2.	Abaanan	7	35%	5	25%
3.	Viyaanan	20	100%	20	100%
4.	Uthaanan	-	-	-	-
5.	Samaanan	20	100%	20	100%
6.	Naagan	-	-	-	-
7.	Koorman	2	10%	-	-
8.	Kirukaran	6	30%	5	25%
9.	Devathathan	20	100%	20	100%
10.	Dhananjayan	-	-	-	-

FIGURE-20 (a)

CONDITION OF VATHAM



From the above table-20 a it is observed that among 20 Out patients 100% were affected in Viyaanan, Samaanan and Devathathan; 35% were affected in Abaanan; 10% were affected in Koorman; 30% were affected in Kirukaran; Among 20 In patients 100% were affected in Viyaanan, Samaanan and Devathathan; 25% were affected in Abaanan.

20 (b). PITHAM

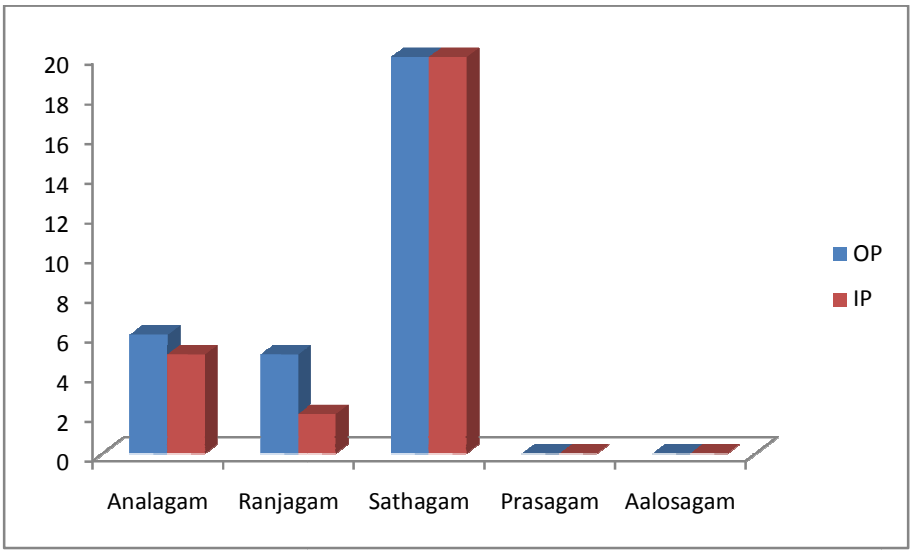
Table-20 (b) illustrates the condition of pitham and its percentage.

TABLE-20 (b) CONDITION OF PITHAM

Sl. No.	Condition of Pitham	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Analagam	6	30%	5	25%
2.	Ranjagam	5	25%	2	10%
3.	Sathagam	20	100%	20	100%
4.	Prasagam	-	-	-	-
5.	Aalosagam	-	-	-	-

FIGURE-20 (b)

CONDITION OF PITHAM



From the above table-20 b it is observed that among 20 Out patients, 100% were affected in Sathagam; 30% were affected in Analagam; 25% were affected in Ranjagam; Among 20 In patients, 100% were affected in Sathagam; 25% were affected in Analagam; 10% were affected in Ranjagam.

20 (c).KAPAM

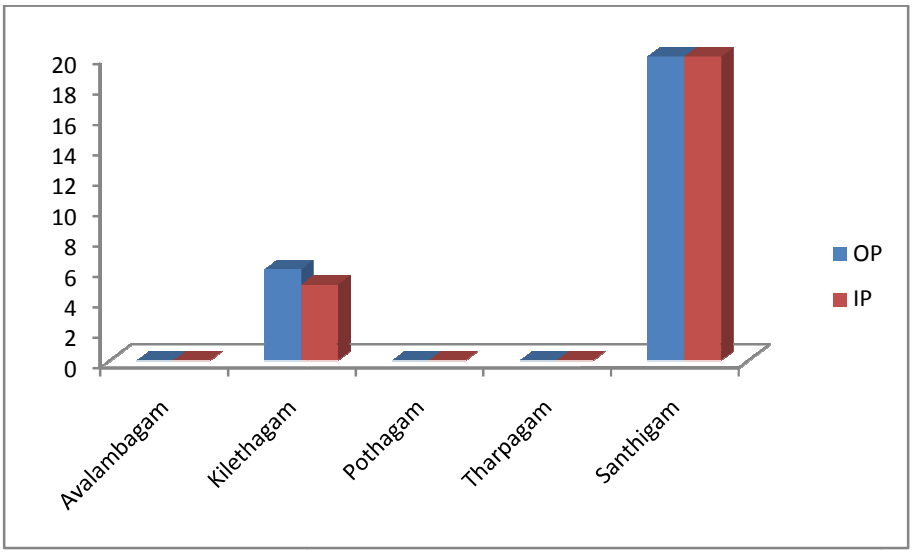
Table-20 (c) illustrates the condition of Kapam and its percentage.

TABLE-20 (c) CONDITION OF KAPAM

Sl. No.	Condition of Kapam	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Avalambagam	-	-	-	-
2.	Kilethagam	6	30%	5	25%
3.	Pothagam	-	-	-	-
4.	Tharpagam	-	-	-	-
5.	Santhigam	20	100%	20	100%

FIGURE-20 (c)

CONDITION OF KAPAM



From the above table-20 c it is observed that among 20 Out patients, 100% were affected in Santhigam; 30% were affected in Kilethagam. Among 20 Inpatients, 100% were affected in Santhigam; 25% were affected inKilethagam.

21. INVOLVEMENT OF UDAL THATHUKKAL

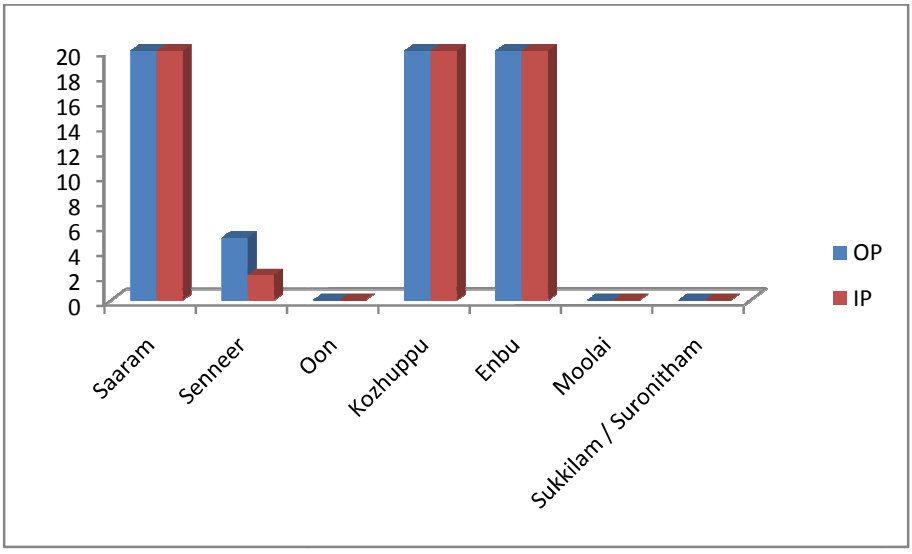
Table-21 illustrates the involvement of udal thathukkal and its percentage.

TABLE-21 INVOLVEMENT OF UDAL THATHUKKAL

Sl. No.	Involvement of Udal Thathukkal	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Saaram	20	100%	20	100%
2.	Senneer	5	25%	2	10%
3.	Oon	-	-	-	-
4.	Kozhuppu	20	100%	20	100%
5.	Enbu	20	100%	20	100%
6.	Moolai	-	-	-	-
7.	Sukkilam / Suronitham	-	-	-	-

FIGURE-21

INVOLVEMENT OF UDAL THATHUKKAL



From the above table-21 it is observed that among 20 Out patients and 20 In patients cent per cent were affected in Saaram, Kozhuppu, Enbu. Among 20 Out patients, 25% were affected in Senneer and in 20 In patients, 10% were affected in Senneer.

22. CONDITIONS OF ENVAGAI THERVUGAL

Table-22 illustrates the envagai thervugal and and its percentage.

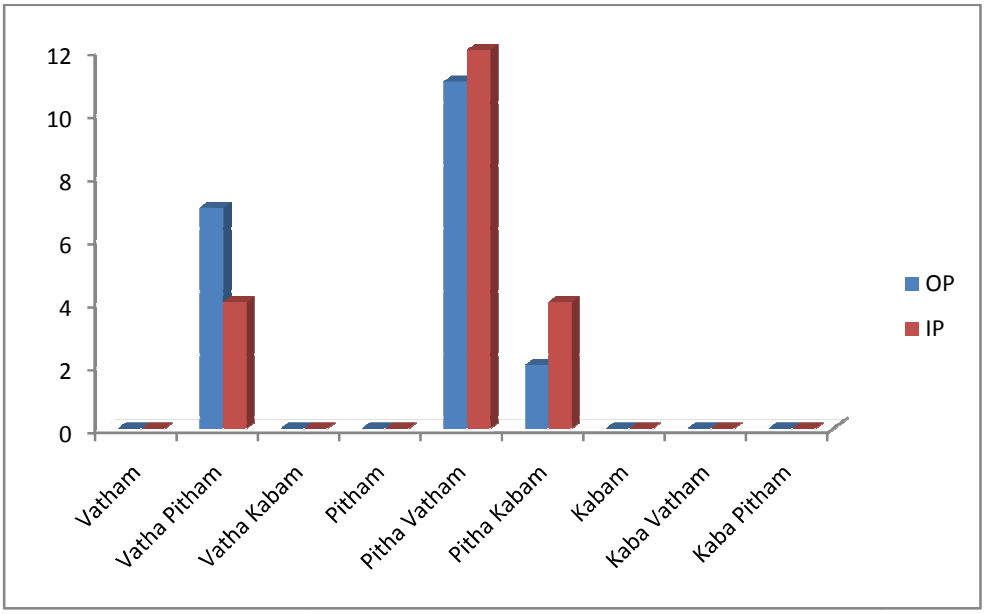
TABLE-22

CONDITIONS OF ENVAGAI THERVUGAL

Sl. No.	Conditions of Envagai Thervugal	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Naadi (Thontha Naadi)				
	1). Vatha Pitham	7	35%	4	20%
	2). Vatha Kapam	-	-	-	-
	3). Pitha Vatham	11	55%	12	60%
	4). Pitha Kapam	2	10%	4	20%
	5). Kaba Vatham	-	-	-	-
	6). Kaba Pitham	-	-	-	-
2.	Sparisam	5	25%	6	30%
3.	Naa	-	-	-	-
4.	Niram	-	-	-	-
5.	Mozhi	-	-	-	-
6.	Vizhi	-	-	-	-
7.	Malam	7	35%	5	25%
8.	Moothiram	-	-	-	-

FIGURE-22

CONDITIONS OF ENVAGAI THERVUGAL



From the above table-22 it is observed that among 20 Out patients, 35% have Vatha Pitha Naadi, 55% have Pitha Vatha Naadi and 10% have Pitha Kaba Naadi; 25% were affected in Sparisam; 35% were affected in Malam. Among 20 In patients, 20% have Vatha Pitha Naadi, 60% have Pitha Vatha Naadi and 20% have Pitha Kaba Naadi ; 30% were affected in Sparisam; 25% were affected inMalam.

23. NEERKURI

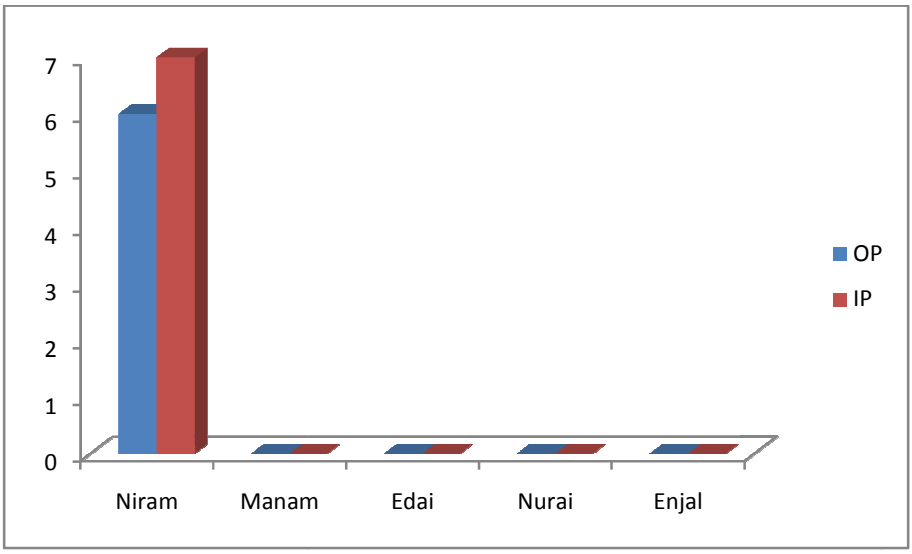
Table-23 illustrates the neer kuri and its percentage.

TABLE-23 NEERKURI

Sl. No.	Neer Kuri	Out Patients (OP)		In Patients (IP)	
		No.of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Niram	6	30%	7	35%
2.	Manam	-	-	-	-
3.	Edai	-	-	-	-
4.	Nurai	-	-	-	-
5.	Enjal	-	-	-	-

FIGURE-23

NEERKURI



From the above table-23 it is observed that among 20 Out patients, 30% were affected in Niram. Among 20 In patients, 35% were affected in Niram.

24. NEIKURI

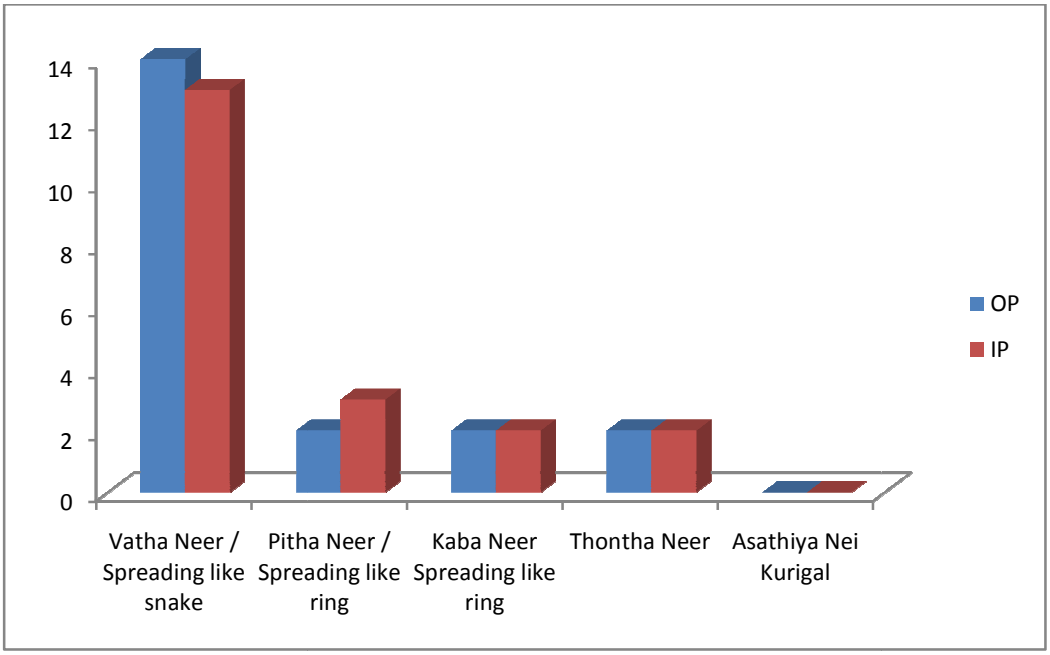
Table-24 illustrates the neikuri and its percentage.

TABLE-24 NEIKURI

Sl. No.	Nei Kuri	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Vatha Neer / Spreading like snake	14	70%	13	65%
2.	Pitha Neer / Spreading like ring	2	10%	3	15%
3.	Kapa Neer / Spreading like pearl	2	10%	2	10%
4.	Thontha Neer	2	10%	2	10%
5.	Asathiya Nei Kurigal	-	-	-	-

FIGURE-24

NEIKURI



From the above table-24 it is observed that among 20 Out patients, 70% have Vatha Neer; 10% Pitha Neer; 10% Kapa Neer and 10% Thontha Neer. Among 20 In patiens, 65% have Vatha Neer; 15% Pitha Neer; 10% Kaba Neer and 10% Thontha Neer.

25. RADIOLOGICAL FINDINGS

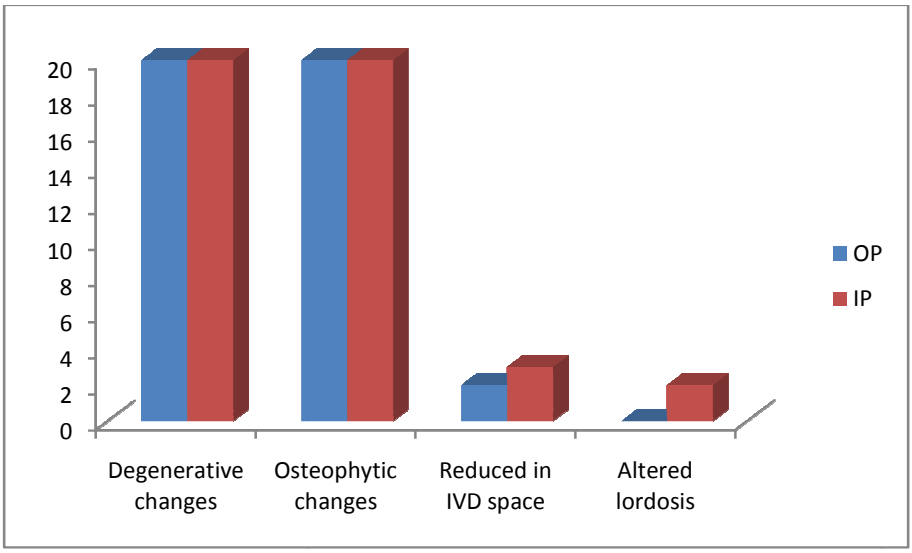
Table-25 illustrates radiological findings and its percentage.

TABLE-25 RADIOLOGICAL FINDINGS

Sl. No.	Radiological Findings	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Degenerative changes	20	100%	20	100%
2.	Osteophytic changes	20	100%	20	100%
3.	Reduced in IVD space	2	10%	3	15%
4.	Altered lordosis	-	-	2	10%

FIGURE-25

RADILOGICAL FINDINGS



From the above table-25 it is observed that among 20 Out patients and 20 In patients, cent percent have degenerative and osteophytic changes. Among 20 Out patients, 10% have reduced IVD space. Among 20 In patients, 15% have reduced IVD space; 10% have reduced lordosis.

26. ASSESSMENT OF OUTCOME

(a) Cardinal Signs

Table-26 (a) (i) illustrates assessment of out come by cardinal signs (Out patients).

TABLE-26 (a) (i)

CARDINAL SIGNS ASSESSMENT-OUT PATIENTS

(n=20) (List wise)

Sl. No.	Cardinal Signs	Out patients						
		Before Treatment	After Treatment	S.D	S.E.	T	P	Result
1.	Low back pain	2.75	0.65	1.23	0.35	5.92	<0.001	Improved
2.	Radiating pain	2.25	0.75	1.21	0.21	7.09	<0.001	Improved
3.	Restricted movements	1.20	0.55	0.94	0.20	3.11	<0.01	Improved
4.	Stiffness	0.90	0.60	0.99	0.12	2.34	<0.05	Improved
5.	Tenderness	0.90	0.45	0.69	0.11	3.94	<0.001	Improved
6.	Numbness	2.55	2.85	0.37	0.10	2.85	<0.05	Improved

Improved

Among 20 Out patients, 100% had the history of low back pain; 40% had radiatingpainofGrade2,35%ofGrade3&10%ofGrade4;10%hadrestrictedMovementsof Grade4,15%ofGrade3,15%ofGrade2,5%ofGrade1;20%hadstiffness of Grade 3, 15 % of Grade 2; 5% had tenderness of Grade 3, 30% of Grade2& 15% of Grade 1; 15% of numbness of Grade 2 & 15% of Grade 1. After treatment, the results are statistically Improved at various P values.

Table-26 (a) (ii) illustrates assessment of out come by cardinal signs (In patients).

TABLE-26 (a) (ii)

CARDINAL SIGNS ASSESSMENT-IN PATIENTS

(n=20) (List wise)

Sl. No.	Cardinal Signs	In patients						
		Before Treatment	After Treatment	S.D	S.E.	T	P	Result
1.	Low back pain	3.00	0.50	0.95	0.22	11.18	<0.001	Improved
2.	Radiating pain	2.80	0.80	1.32	0.27	7.36	<0.001	Improved
3.	Restricted movements	1.40	0.50	0.95	0.20	4.41	<0.001	Improved
4.	Stiffness	1.20	0.60	1.05	0.19	3.04	<0.01	Improved
5.	Tenderness	0.45	0.25	0.64	0.09	2.17	<0.05	Improved
6.	Numbness	2.50	2.85	0.37	0.10	3.19	<0.05	Improved

Improved

Among 20 In patients, 100% had the history of low back pain; 55% had radiating pain of Grade2, 20% of Grade3 & 15% of Grade4; 10% of Grade1; 20% had restricted movements of Grade3, 15% of Grade2, 10% of Grade4, 10% of Grade1; 10% had stiffness of Grade 3, 10 % of Grade 2; 10% of Grade 1; 10% of had tenderness of Grade 1, 10% of Grade 2, 5% of Grade 3; 20% had numbness of Grade 2 & 15% of Grade 1. After treatment, the results are statistically Improved at various Pvalues.

Table-26 (b) (i) illustrates assessment of out come by range of motion (Out patients).

TABLE-26 (b) (i)

RANGE OF MOTION-OUT PATIENTS

(n=20) (List wise)

Sl. No.	Range of Motion	Out patients						
		Before Treatment	After Treatment	S.D	S.E.	T	P	Result
1.	Flexion	0.55	1.70	0.47	0.08	14.03	<0.001	Improved
2.	Extension	0.50	1.65	0.59	0.10	10.50	<0.001	Improved
3.	Rotation	0.30	1.65	0.49	0.20	6.46	<0.001	Improved
4.	Lateral flexion	0.32	1.50	0.51	0.09	12.59	<0.001	Improved

Among 20 Out patients, 100% of cases has history of difficulty in the range of movements. Flexion-30% with Grade 1 & 70% with Grade 0, Extension-35% with Grade 1 & 65% with Grade 0.Rotation-55% with Grade 1 & 45% with Grade 0, Lateral flexion-30% with Grade 1 & 70% with Grade 0. After treatment, the results are satistically was ImprovedatP<0.001.

Table-26 (b) (ii) illustrates assessment of out come by range of motion (In patients).

TABLE-26 (b) (ii)

RANGE OF MOTION-IN PATIENTS

(n=20) (List wise)

Sl. No.	Range of motion	In patients						
		Before Treatment	After Treatment	S.D	S.E.	T	P	Result
1.	Flexion	0.50	1.30	0.57	0.09	8.71	<0.001	Improved
2.	Extension	0.55	1.35	0.67	0.09	8.71	<0.001	Improved
3.	Rotation	0.50	1.20	0.77	0.10	6.65	<0.001	Improved
4.	Lateral flexion	0.45	1.10	0.31	0.13	4.95	<0.001	Improved

Among 20 Out patients, 100% had the difficulty in active range of movements. Flexion-50% with Grade 1 & 50% with Grade 0, Extension-55% with Grade 1 & 45% with Grade 0, Rotation-50% with Grade 1 & 50% with Grade 0, Lateral flexion-45% with Grade 1 & 55% with Grade 0. After treatment the results are statistically

Improved atP<0.001.

26. (c) (i) Back Pain Functional Scale Score

Table-26 (c) (i) illustrates back pain functional scale score in percentage.

TABLE-26 (c) (i)

BACK PAIN FUNCTIONAL SCORE SCALE

Sl. No.	Assessment of Outcome (Pain Score)	Before Treatment				After Treatment			
		Out Patients (OP)		In Patients (IP)		Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)	No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	≤30	20	100%	20	100%	2	10%	2	10%
2.	31-40	-	-	-	-	2	10%	4	20%
3.	41-50	-	-	-	-	3	15%	-	-
4.	51-60	-	-	-	-	13	65%	14	70%

Minimum Score-0, Maximum Score-60

Pain Score	Improvement
≤30	NO
31-40	Mild
41-50	Moderate
51-60	Good

Reference:

Stratford PW Binkley JM et al. Development and initial validation of the Back pain Functional Scale. Spine.2000; 2095-2102 (Appendix-A Page: 2101)

From the above table, it is observed that before treatment among 20 Out patients and 20 In patients cent percent were with pain score ≤30. After treatment, among 20 Out patients 65% have good improvement; 15%: moderate imporevement; 10% mild improvement; 10% no improvement. Among 20 In patient 70% have good improvement; 20% mild improvement; 10% no improvement.

FIGURE-26 (c) (i)

BACK PAIN FUNCTIONAL SCALE SCORE

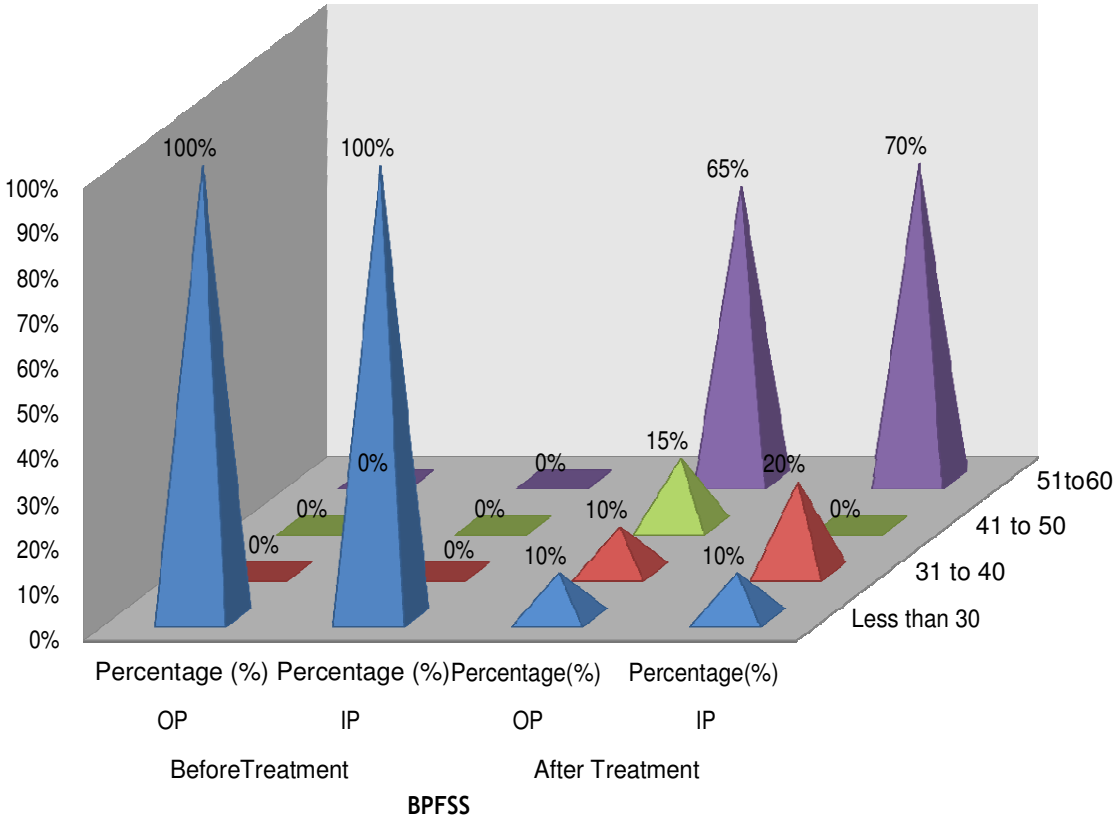


Table-26 (c) (ii) illustrates BPFSS-Out Patients.

TABLE-26 (c) (ii) BPFSS-OUT PATIENTS

Sl. No.	Out patients	N	Minimum	Maximum	Mean	SD	SEM
1.	Before treatment	20	10.00	30.00	21.85	5.40	1.21
2.	After treatment	20	24.00	59.00	48.20	10.14	2.27
	N (list wise)	20					

Correlations

			Before treatment	After Treatment
1.	Before treatment	Pearson correlations	1	0.821**
		Sig. (2tailed)		0.000
		N	20	20
2.	After treatment	Pearson correlations	0.821**	1
		Sig. (2tailed)	0.000	
		N	20	20

***Correlation is significant at the 0.01 level (2-tailed)*

Pearson correlation (γ) before and after treatment is 0.821. One way analysis if variance ANOVA; p-value followed to be less than 0.001 is considered to be extremely statisticallysignificant.There is strong evidence (t=18.524, P<0.001) that the clinical trial drug improves the Thandaga Vatham patients. In this data set, we could get a mean paired difference - 26.35, df=19 with the confidence interval of 95% the null hypothesis is rejected, since P<0.001. The relation is positive which means that as one variable goes up or down so will the otherone.

Table-26 (c) (iii) illustrates BPFSS-In Patients.

TABLE-26 (c) (iii) BPFSS-INPATIENTS

(n=20) (List wise)

Sl. No.	In patients	N	Minimum	Maximum	Mean	SD	SEM
1.	Before treatment	20	10.00	28.00	19.10	5.34	1.20
2.	After treatment	20	17.00	58.00	46.50	12.26	2.74

Correlations

(n=20) (List wise)

			Before treatment	After Treatment
1.	Before treatment	Pearson correlations	1	0.413**
		Sig. (2tailed)		0.000
		N	20	20
2.	After treatment	Pearson correlations	0.413**	1
		Sig. (2tailed)	0.000	
		N	20	20

***Correlation is significant at the 0.01 level (2-tailed)*

Pearson correlation (γ) before and after treatment among In patient is 0.413. One way analysis if variance ANOVA; p-value followed to be less than 0.0001 is considered to be extremely statistically significant.

There is strong evidence (t=10.9651, P<0.001) that the clinical trial drug improves the patients. Mean paired difference-27.10,df=19 with 95% confidence interval. The null hypothesis is rejected, since P<0.001 suggesting the relationship is a positive.

27. GRADATION OFRESULTS

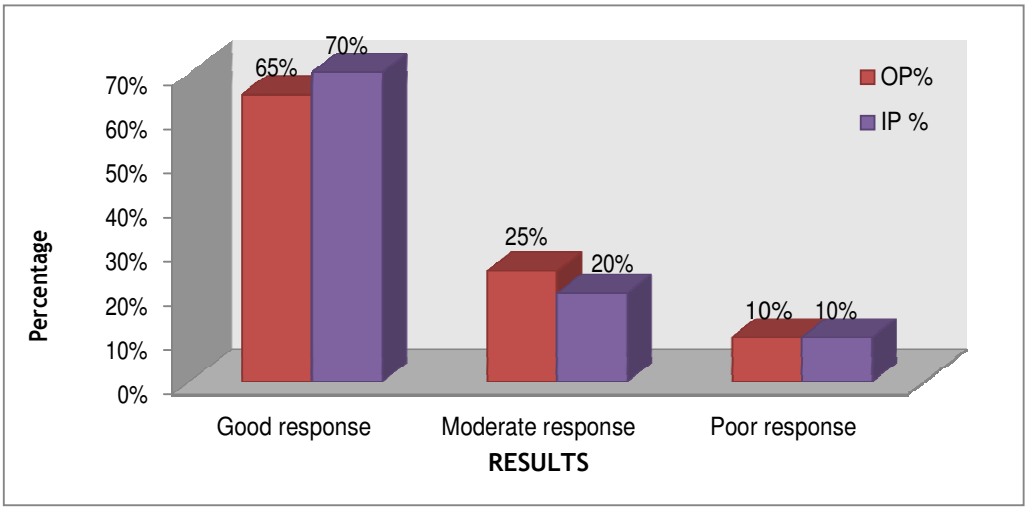
Table-27 illustrates Gradation of Results and its percentage.

TABLE-27 GRADATION OF RESULTS

Sl. No.	Results	Out Patients (OP)		In Patients (IP)	
		No. of cases	Percentage (%)	No. of cases	Percentage (%)
1.	Good response	13	65%	14	70%
2.	Moderate response	5	25%	4	20%
3.	Poor response	2	10%	2	10%
	Total	**20**	**100%**	**20**	**100%**

FIGURE-27

GRADATION OF RESULTS



From the above table, it is observed that among 20 Out patients, 65% showed good response; 25% moderate response; 10% poor response. Among 20 In patients, 70% showed good response; 20% moderate response; 10% poor response.

TABLE-28 (a)
LABORATORY INVESTIGATION (OUT PATIENTS)

Sl. No.	Out Patient	Haematological Report														Urine Analysis					
		Before Treatment							AfterTreatment							Before Treatment			After Treatment		
		TC	DC			ESR		Hb% (gms)	TC	DC			ESR		Hb% (gms)	Alb.	Sug.	Dep- Epi. Cells / Puscells	Alb.	Sug.	Dep- Epi. Cells/ Puscells
		Cells / cu mm	P	L	E	½ Hr.	1 Hr.		Cells / cu mm	P	L	E	½ Hr.	1 Hr.							
1.	34781	6800	60	37	3	10	24	8.5	7000	61	34	5	9	15	9.1	NIL	NIL	2-3PUS CELLS	NIL	NIL	NAD
2.	35245	9400	68	23	7	3	15	10.5	9700	71	23	6	2	14	11.2	NIL	NIL	NAD	NIL	NIL	NAD
3.	35281	9100	66	32	6	4	9	12.4	9400	65	29	6	5	10	12.8	NIL	NIL	NAD	NIL	NIL	NAD
4.	35574	5900	56	38	6	15	32	9.4	6000	62	34	4	3	5	10.5	NIL	NIL	1-3 EPI CELLS	NIL	NIL	NAD
5.	40506	8400	64	36	5	3	7	12.2	8600	65	30	7	3	6	12.4	NIL	NIL	NAD	NIL	NIL	NAD
6.	42877	8200	66	30	4	4	12	10.7	8400	67	30	5	3	9	11.2	NIL	NIL	NAD	NIL	NIL	NAD
7.	46407	8300	70	24	3	7	16	12.8	8600	72	24	4	8	14	13.0	NIL	NIL	NAD	NIL	NIL	NAD
8.	47318	9300	64	38	2	9	15	11.2	9200	61	36	3	8	12	11.5	NIL	NIL	NAD	NIL	NIL	NAD
9.	48538	8200	66	28	4	8	12	12	8400	68	29	5	9	12	12.3	NIL	NIL	1-2PUS CELLS	NIL	NIL	NAD
10.	51412	8900	64	32	4	9	17	12.5	9000	66	31	3	8	16	12.3	NIL	NIL	NAD	NIL	NIL	NAD
11.	62571	9400	66	30	4	5	15	11.0	9600	67	31	2	4	13	11.10	NIL	NIL	NAD	NIL	NIL	NAD
12.	63624	9500	60	34	6	4	14	11.0	9400	62	33	5	3	9	11.4	NIL	NIL	NAD	NIL	NIL	NAD
13.	64180	7800	62	34	6	4	8	11.8	7900	64	33	6	3	6	12.2	NIL	NIL	NAD	NIL	NIL	NAD
14.	70364	9200	60	38	2	2	4	12.6	9300	63	34	3	2	4	12.7	NIL	NIL	1-2 PUS CELLS	NIL	NIL	NAD
15.	106698	8600	70	26	4	9	14	12.2	8700	71	24	5	7	12	12.5	NIL	NIL	NAD	NIL	NIL	NAD
16.	108637	8400	62	36	2	7	14	12.5	8900	62	35	3	5	11	13.1	NIL	NIL	NAD	NIL	NIL	NAD
17.	3316	8800	62	33	7	3	9	10.9	9000	61	33	6	2	8	11.5	NIL	NIL	NAD	NIL	NIL	NAD
18.	4418	6900	64	34	2	5	15	13.4	7000	65	32	3	4	12	13.6	NIL	NIL	NAD	NIL	NIL	NAD
19.	7908	8900	61	36	3	9	15	10.1	8800	62	34	4	8	13	11.0	NIL	NIL	1-3EPI CELLS	NIL	NIL	NAD
20.	9165	7600	62	30	6	7	17	11.0	7900	65	30	5	6	14	11.4	NIL	NIL	NAD	NIL	NIL	NAD

TABLE-28 (b)

LABORATORY INVESTIGATION (IN PATIENTS)

Sl. No.	In Patient No.	Haematological Report														Urine Analysis					
		Before Treatment							AfterTreatment							Before Treatment			After Treatment		
		TC	DC			ESR		Hb% (gms)	TC	DC			ESR		Hb% (gms)	Alb.	Sug.	Dep- Epi. Cells / Puscells	Alb.	Sug.	Dep- Epi. Cells/ Puscells
		Cells / cu mm	P	L	E	½ Hr.	1 Hr.		Cells / cu mm	P	L	E	½ Hr.	1 Hr.							
1.	1503	7000	54	44	2	2	6	10.2	7200	60	38	2	2	4	11.2	NIL	NIL	NAD	NIL	NIL	NAD
2.	1588	8900	58	34	8	6	9	13.0	9000	62	32	6	4	8	13.2	NIL	NIL	NAD	NIL	NIL	NAD
3.	1794	10200	65	30	5	9	14	13.4	10000	64	32	4	7	12	13.3	NIL	NIL	NAD	NIL	NIL	NAD
4.	1811	9600	72	26	2	10	20	10.7	9800	73	24	2	8	16	11.3	NIL	NIL	1-2 Pus Cells	NIL	NIL	NAD
5.	1892	8600	60	37	3	9	20	11.5	9200	65	33	2	3	6	12.2	NIL	NIL	1-2 Pus Cells	NIL	NIL	NAD
6.	1956	9800	70	21	9	6	12	12.9	8400	63	35	2	6	12	13.2	NIL	NIL	NAD	NIL	NIL	NAD
7.	2042	9100	79	20	1	8	20	13.2	9300	79	22	1	6	14	13.2	NIL	NIL	NAD	NIL	NIL	NAD
8.	2043	6500	67	30	3	6	16	13.5	6800	68	33	2	3	6	13.5	NIL	NIL	NAD	NIL	NIL	NAD
9.	2784	9000	69	23	8	8	13	12.4	9100	70	26	4	6	10	12.6	NIL	NIL	NAD	NIL	NIL	NAD
10.	2799	7500	65	31	4	3	6	12.9	8000	63	33	4	4	8	12.8	NIL	NIL	NAD	NIL	NIL	NAD
11.	2832	9100	57	40	3	4	8	11.9	9300	59	39	2	3	7	12.0	NIL	NIL	NAD	NIL	NIL	NAD
12.	2845	6800	63	30	7	9	15	13.0	7000	64	35	1	5	13	12.9	NIL	NIL	1-2Epi Cells	NIL	NIL	NAD
13.	3079	7800	69	24	7	8	12	10.8	7900	65	30	5	6	11	10.8	NIL	NIL	NAD	NIL	NIL	NAD
14.	3142	8900	69	29	2	4	9	12.4	9000	67	36	1	2	6	12.8	NIL	NIL	NAD	NIL	NIL	NAD
15.	87	5900	50	49	1	2	4	10.2	6500	54	45	1	2	4	11.4	NIL	NIL	NAD	NIL	NIL	NAD
16.	101	7400	59	37	4	7	15	11.9	7800	59	38	3	4	8	12.2	NIL	NIL	NAD	NIL	NIL	NAD
17.	110	8400	65	33	2	4	10	10.3	8600	67	27	1	3	10	10.8	NIL	NIL	NAD	NIL	NIL	NAD
18.	118	7800	71	23	6	2	6	14.2	8100	71	35	6	2	6	14.2	NIL	NIL	NAD	NIL	NIL	NAD
19.	126	8800	79	13	8	9	18	14.1	9000	72	29	4	4	9	14.1	NIL	NIL	NAD	NIL	NIL	NAD
20.	504	7100	67	27	6	10	20	13.0	7400	64	27	2	6	11	13.3	NIL	NIL	NAD	NIL	NIL	NAD

TABLE-29 (a)

LABORATORY INVESTIGATION (OUT PATIENTS)

Sl. No.	OP No.	Before Treatment					After Treatment				
		Blood Sugar (R)	Blood Urea	Serum cholesterol	Serum Uric acid	Serum Creatinine	Blood Sugar (R)	Blood Urea	Serum cholesterol	Serum Uric acid	Serum Creatinine
1.	34781	101	32	160	4.00	0.80	100	29	146	3.98	0.70
2.	35245	102	29	141	4.00	1.00	98	27	137	4.10	0.80
3.	35281	120	27	169	3.00	0.80	115	27	166	3.00	0.80
4.	35574	91	22	145	3.50	0.50	134	19	128	3.47	0.70
5.	40506	129	24	119	3.00	0.60	86	25	122	3.10	0.50
6.	42877	101	19	147	3.70	0.80	100	18	140	3.70	0.70
7.	46407	129	25	187	4.20	0.90	117	26	189	4.20	0.70
8.	47318	89	24	190	4.30	0.50	80	23	182	4.30	0.50
9.	48538	94	23	132	2.50	0.80	85	21	140	2.49	0.80
10.	51412	82	22	179	4.40	0.50	121	20	183	4.35	0.60
11.	62571	96	21	170	3.00	0.90	116	22	168	2.99	0.80
12.	63624	91	18	165	3.50	0.40	101	16	160	3.49	0.40
13.	64180	90	20	164	3.60	0.70	86	18	154	3.58	0.70
14.	70364	114	21	189	3.24	0.70	109	19	178	3.21	0.60
15.	106698	90	20	146	3.40	0.60	122	20	161	3.39	0.70
16.	108637	75	25	118	5.00	1.00	99	23	128	4.89	0.90
17.	3316	94	32	163	4.52	0.80	132	30	165	4.47	0.80
18.	4418	112	24	109	3.80	0.80	105	25	107	3.81	0.90
19.	7908	126	24	178	3.20	0.90	99	26	176	3.00	0.80
20	9165	98	30	189	3.00	1.00	116	27	185	3.10	0.70

TABLE-29 (b)

LABORATORY INVESTIGATION (IN PATIENTS)

Sl. No.	IP No.	Before Treatment					After Treatment				
		Blood Sugar (R)	Blood Urea	Serum cholesterol	Serum Uric acid	Serum Creatinine	Blood Sugar (R)	Blood Urea	Serum cholesterol	Serum Uric acid	Serum Creatinine
1.	1503	87	37	197	5.20	1.00	82	32	180	5.00	0.50
2.	1588	105	26	160	3.80	0.60	95	22	158	3.50	0.70
3.	1794	93	28	193	4.60	0.70	114	26	186	4.50	0.80
4.	1811	70	32	140	4.00	0.90	70	30	136	3.80	1.00
5.	1892	105	19	120	2.20	0.80	136	21	118	2.20	0.70
6.	1956	120	24	188	3.00	1.00	102	25	174	3.10	0.80
7.	2042	72	23	175	2.80	0.60	70	24	163	2.70	0.50
8.	2043	86	27	163	4.50	0.60	80	24	177	4.60	0.50
9.	2784	96	21	180	3.60	0.80	92	20	176	3.50	0.60
10.	2799	105	26	200	4.40	0.50	111	24	197	4.60	0.40
11.	2832	103	20	189	5.20	0.20	125	23	182	5.50	0.30
12.	2845	133	30	185	4.30	0.60	91	32	180	4.70	0.70
13.	3079	109	33	192	4.10	0.80	117	31	194	4.60	0.90
14.	3142	85	19	156	3.60	0.80	83	18	149	3.40	0.60
15.	87	99	17	142	3.90	1.00	97	20	155	3.20	0.90
16.	101	111	27	164	4.00	0.70	80	24	170	3.80	0.80
17.	110	92	15	200	3.90	0.50	92	17	196	4.10	0.40
18.	118	102	30	130	4.30	0.80	100	27	124	4.20	0.70
19.	126	78	24	198	2.40	0.90	80	26	193	2.20	0.90
20	504	97	19	181	2.20	0.40	133	18	177	2.60	0.80

TABLE-30 (a)

LABORATORY AND RADIOLOGICAL INVESTIGATIONS (OUT PATIENTS)

Sl. No.	OP No.	RA Factor (IU/ ml)	ASO titre (IU/ ml)	C-reative protin (mg/dl)	Radiological finding
1	34781	16.7	76.4	4.9	Spondylotic changes L2 to L4
2	35245	18.1	64.0	1.8	Spondylotic changes L1 to L4
3	35281	19.0	49	5.5	Spondylotic changes L3 to L5
4	35574	14.1	90.1	4.7	Spondylotic changes L3 to L4
5.	40506	10.9	87.3	3.5	Spondylotic changes L3 to L5
6	42877	12.3	79.8	2.6	Spondylotic changes L5 to S1
7	46407	17.5	80.7	2.8	Spondylotic changes L3 to L5
8	47318	17	79.7	2.5	Spondylotic changes L2 to L5
9	48538	19.1	88.4	2.2	Spondylotic changes L4 to S1
10	51412	6.6	90.3	1.6	Spondylotic changes L1 to L4
11	62571	9.3	91.2	2.3	Spondylotic changes L3 to L4
12	63624	8.1	74.0	3.4	Spondylotic changes L5 to S1
13	64180	10	93.0	5.4	Spondylotic changes L4 to L5
14	70364	16.3	100.0	4.5	Spondylotic changes L2 to L5
15	106698	15.6	88.0	2.7	Spondylotic changes L3 to L5
16	108637	12.4	90.7	1.6	Spondylotic changes L3 to L5
17	3316	16	111.0	2.9	Spondylotic changes L3 to L5
18	4418	18.4	80.1	4.0	Spondylotic changes L2 to L4
19	7908	13.7	104.0	3.1	Spondylotic changes L3 to L5
20	9165	12.6	91.2	4.2	Spondylotic changes L3 to L5

Reference Range:

Rheumatoid Factor: Upto 20 IU/ml Serum for ASO: Upto 200IU/ml CRP: Upto 6 mg/l

TABLE-30 (b)

LABORATORY AND RADIOLOGICAL INVESTIGATIONS (IN PATIENTS)

Sl. No.	IP No.	RA Factor (IU/ ml)	ASO titre (IU/ ml)	C-reative protin (mg/dl)	Radiological finding
1.	1503	7.4	100.9	2.9	Spondylotic changes L4 to L5
2.	1588	10.4	69.9	4.5	Spondylotic changes L3 to L5
3.	1794	15.7	80.0	3.0	Spondylotic changes L3 to L5
4.	1811	1.30	40.0	4.0	Spondylotic changes L2 to L5
5.	1892	7.0	79.8	5.0	Spondylotic changes L3 to L4
6.	1956	9.2	61.1	3.2	Spondylotic changes L3 to L5
7.	2042	5.6	90.2	5.3	Spondylotic changes L3 to L4
8.	2043	13.1	76.8	2.4	Spondylotic changes L3 to L5
9.	2784	6.9	69.5	3.1	Spondylotic changes L3 to L5
10.	2799	4.1	71.0	6.5	Spondylotic changes L3 to L5
11.	2832	6.3	11.0	4.8	Spondylotic changes L5 to S1
12.	2845	6.9	60.8	5.2	Spondylotic changes L1 to L5
13.	3079	6.6	20.0	1.8	Spondylotic changes L3 to L5
14.	3142	7.8	81.6	4.6	Spondylotic changes L3 to L4
15.	87	3.8	79.6	3.8	Spondylotic changes L4 to L5
16.	101	5.4	101.4	2.7	Spondylotic changes L4 to L5
17.	110	4.5	80.9	5.5	Spondylotic changes L4 to S1
18.	118	7.9	70.2	3.5	Spondylotic changes L4 to L5
19.	126	5.2	55.8	2.9	Spondylotic changes L3 to L5
20	504	8.2	93.6	4.2	Spondylotic changes L3 to L4

Reference range:

Rheumatoid Factor: Upto 20IU/ml Serum for ASO: Upto 200 IU/ml CRP: Upto 6 mg/L

TABLE-31 (a)

BACK PAIN FUNCTIONAL SCALE SCORE VALUES (OUT PATIENTS)

Sl. No.	OP No.	Pain score	
		Before Treatment	After Treatment
1.	34781	30	52
2.	35245	27	55
3.	35281	26	57
4.	35574	28	51
5.	40506	22	55
6.	42877	25	53
7.	46407	21	59
8.	47318	19	45
9.	48538	27	54
10.	51412	25	58
11.	62571	23	52
12.	63624	13	33
13.	64180	16	39
14.	70364	23	53
15.	106698	26	51
16.	108637	20	43
17.	3316	10	27
18.	4418	15	24
19.	7908	17	47
20	9165	24	56

Interpretation:

≤30 : No improvement / High disease activity

31-40 : Mild improvement

41-50 : Moderate improvement

51-60 : Good improvement

TABLE-31 (b)

BACK PAIN FUNCTIONAL SCALE SCORE VALUES (IN PATIENTS)

Sl. No.	IP No.	Pain score	
		Before Treatment	After Treatment
1.	1503	28	51
2.	1588	17	32
3.	1794	24	53
4.	1811	22	56
5.	1892	23	53
6.	1956	19	51
7.	2042	15	54
8.	2043	12	17
9.	2784	11	24
10.	2799	18	52
11.	2832	10	58
12.	2845	16	54
13.	3079	27	56
14.	3142	21	36
15.	87	25	52
16.	101	16	34
17.	110	20	55
18.	118	14	57
19.	126	26	52
20	504	18	33

Interpretation:

≤30 : No improvement / High diseas activity

31-40 : Mild improvement

41-50 : Moderate improvement

51-60 : Good improvement

TABLE-32 (a)

CASE SUMMARY (OUT PATIENTS)

Sl. No.	OP No.	Name	Age / Sex	Occupation	Duration of illness	Treatment starting date	End of treatment	Total Days	Results
1.	34781	Murugammal	42/F	Hose wife	6 Month	16.04.2018	16.05.2018	31	Good
2.	35245	Subbulakshmi	55/F	Hose wife	6 Month	18.04.2018	18.05.2018	31	Good
3.	35281	Narayana vadivu	55/F	Hose wife	3 Years	18.04.2018	19.04.2018	32	Fair
4.	35574	Subbulakshmi	39/F	Hose wife	10 Month	19.04.2018	20.05.2018	32	Good
5.	40506	Jeya Lakshmi	45/F	Coolie	1 Year	07.05.2018	07.06.2018	32	Good
6.	42877	Kali eswari	40/F	Tailor	1 ½ Years	15.05.2018	16.06.2018	32	Poor
7.	46407	Mathiyalagan	54/M	Agriculture Labour	2 Years	29.05.2018	30.06.2018	32	Fair
8.	47318	Sanmuga Nathan	57/M	Business	6 Month	01.06.2018	01.07.2018	31	Good
9.	48538	Lakshmi	56/F	Tailor	1 Year	06.06.2018	05.07.2018	30	Good
10.	51412	Senthil kumar	48/M	Agriculture Labour	8 Month	18.06.2018	17.07.2018	30	Good
11.	62571	Kokila	31/F	House wife	2 Years	27.07.2018	29.08.2018	33	Fair
12.	63624	Murugan	38/M	Coolie	4 Month	31.07.2018	30.08.2018	30	Good
13.	64180	Mariyapan	51/M	Driver	1 ½ Years	02.08.2018	01.09.2018	32	Fair
14.	70364	Rajaguru	30/M	Agricultural labour	6 Month	24.08.2018	23.09.2018	30	Good
15.	106698	Krishnan	58/M	Coolie	1 Years	25.12.2018	26.01.2019	33	Good
16.	108637	Akni muthu	41/M	Agriculture Labour	2 Years	31.12.2018	30.01.2019	31	Fair
17.	3316	Nambi rajan	35/M	Coolie	1 Year	07.01.2019	07.02.2019	31	Good
18.	4418	Muthuvel	46/M	Business	2 Years	10.01.2019	10.02.2019	31	Poor
19.	7908	Mardisudu	46/M	Tailor	3 Month	21.01.2019	19.02.2019	30	Good
20	9165	Sankara narayanan	38/M	Clerk	1 Month	24.01.2019	22.02.2019	30	Good

TABLE-32 (b)

CASE SUMMARY (IN PATIENTS)

Sl. No.	IP No.	Name	Age / Sex	Occupation	Duration of illness	Treatment starting date	End of treatment	Total Days		Total Days	Results
								IP	OP		
1.	1503	Jayapal	69/M	Coolie	1 Month	11.06.2018	12.06.2018	31	-	31	Good
2.	1588	Senthilkumar	48/M	Agricultural labour	1 Month	19.06.2018	18.07.2018	30	-	30	Good
3.	1794	Deivakani	40/F	Housewife	2 Years	16.07.2018	14.08.2018	30	-	30	Poor
4.	1811	Abdulkabur	60/M	Agricultural labour	1 Year	17.07.2018	15.08.2018	30	-	30	Fair
5.	1892	Lakshmi	60/F	Housewife	2 Month	25.07.2018	24.08.2018	31	-	31	Good
6.	1956	Rajaguru	30/M	Agricultural labour	6 Month	01.08.2018	22.08.2018	22	08	30	Good
7.	2042	Mariyapan	51/M	Driver	6 Month	09.08.2018	08.09.2018	30	-	30	Good
8.	2043	Mariselvam	34/M	Coolie	2 Month	09.08.2018	08.09.2018	30	-	30	Good
9.	2784	Selvi	53/F	Housewife	2 Years	15.11.2018	15.12.2018	31	-	31	Poor
10.	2799	Mathiyalagan	56/M	Agricultural labour	1 Month	16.11.2018	15.12.2018	30	-	30	Good
11.	2832	Marimuthu	45/M	Agricultural labour	2 Month	19.11.2018	18.12.2018	30	-	30	Good
12.	2845	Thangam	43/F	Housewife	6 Month	20.11.2018	19.12.2018	30	-	30	Good
13.	3079	Subhiya	36/M	Tailor	1 Year	14.12.2018	12.01.2019	30	-	30	Fair
14.	3142	Sundaramal	60/F	Housewife	6 Month	25.12.2018	23.01.2019	30	-	30	Fair
15.	87	Murugan	60/M	Coolie	3 Month	19.01.2019	17.02.2019	30	-	30	Good
16.	101	Murugan	32/M	Driver	8 Month	21.01.2019	19.02.2019	30	-	30	Fair
17.	110	Murugan	60/M	Agricultural labour	2 Month	21.01.2019	19.02.2019	30	-	30	Good
18.	118	Mani	30/M	Coolie	1 Month	22.01.2019	20.02.2019	30	-	30	Good
19.	126	Umayar	48/M	Coolie	4 Month	24.01.2019	22.02.2019	30	-	30	Fair
20	504	Boopathi raja	56/M	Driver	2 Years	27.02.2019	31.03.2019	33	-	33	Poor

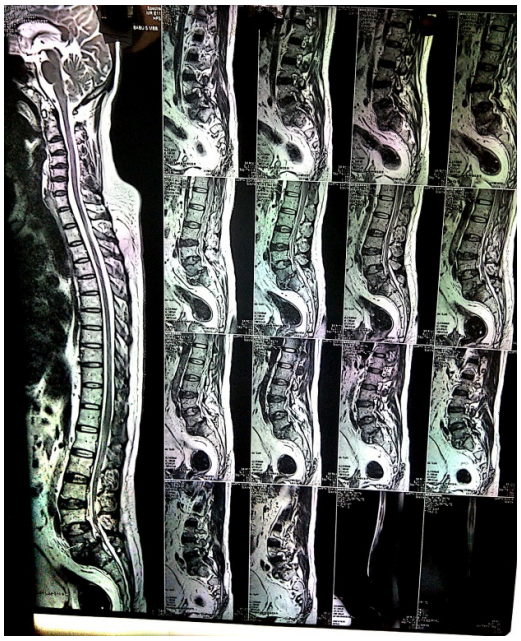
MRI REPORTS

IP No.1588 Patient Name: Mr.**SENTHIL KUMAR**(48/M)

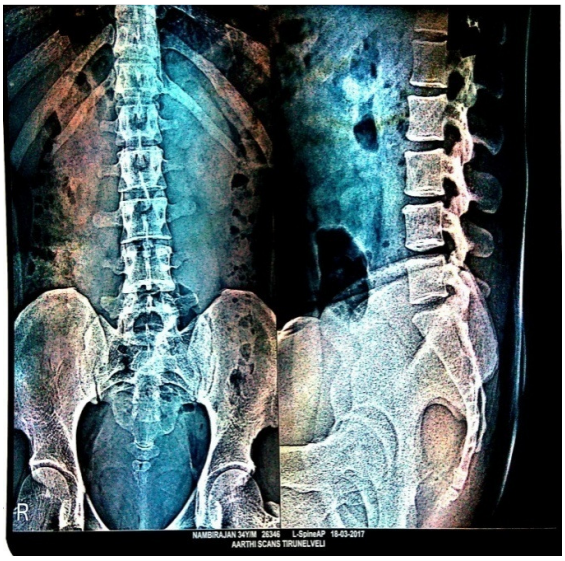
IP No.8073 Patient Name: Mr.**MANI**(30/M)

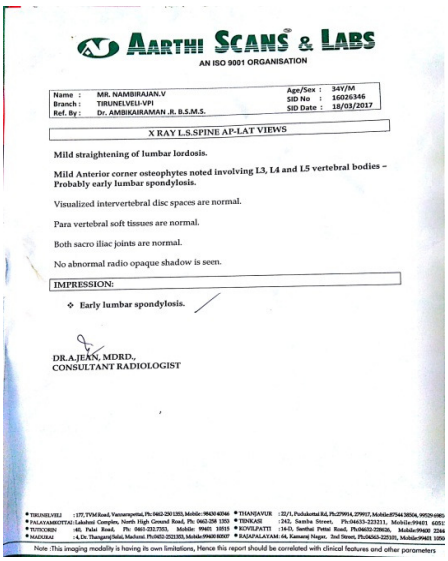
IP No.1503 Patient Name: Mr.**JAYAPAUL**(60/M)

IP No.2042 Patient Name: Mr.**MARIYAPAN**(51/M)



OP No.3316 Patient Name: Mr.**NAMBIRAJAN**(35/M)





AARTHI SCANS & LABS
AN ISO 9001 ORGANISATION

Name : MR. NAMBIRAJAN.V
Branch : TIRUNELVELI-VPI
Ref. By : Dr. AMBIKAIRAMAN .R. B.S.M.S.

Age/Sex : 34Y/M
SID No : 16026346
SID Date : 18/03/2017

X RAY L.S.SPINE AP-LAT VIEWS

Mild straightening of lumbar lordosis.

Mild Anterior corner osteophytes noted involving L3, L4 and L5 vertebral bodies – Probably early lumbar spondylosis.

Visualized intervertebral disc spaces are normal.

Para vertebral soft tissues are normal.

Both sacro iliac joints are normal.

No abnormal radio opaque shadow is seen.

IMPRESSION:

- Early lumbar spondylosis.

DR.A.JEAN, MDRD.,
CONSULTANT RADIOLOGIST

Note :This imaging modality is having its own limitations, Hence this report should be correlated with clinical features and other parameters

IP No.126 Mr. **UMAYAR**(48/M)

IP No.2043 Patient Name: Mr.**MARISELVAM**(34/M)

CHAPTER-VI

DISCUSSION

Thandaga Vatham described by Yugi Munivar in Yugi Vaidhiya Chinthamani-800 is nearly correlated with Lumbar Spondylosis. For this clinical trial study totally 40 patients were selected, 20 were treated as Out patients and 20 were treated as In patients with clinical trial drug **MUNNAI ILAI KUDINEER** 30ml twice a day for 30 days. The most important clinical features of Thandaga Vatham is pain in low back area, stiffness, restricted movements, tenderness, numbness and radiating pain. The diagnosis was made by Siddha and modern diagnostic tools.Institutional ethiclal committee clearance was obtained for this study with **IEC No. (GSMC-IV-IEC/2017/Br-I/07/29.05.2017**).

To evaluate the standardization of the trial drug, it was authenticated through visual inspection and organoleptic characters. To ensure safety is made through *vivo and vitro* studies.

The observed results were discussed below:

1. Incidence with Age Distribution:

The disease was found to be higher in the age group of 51-60 in both OP and IP (OP-35% & IP-50%)

2. Incidence with Sex Distribution:

- 60% of the Out paitents were males and 40% werefemales.
- 75% of the In patients were males and 25% werefemales.

3. Distribution according to Kaalam:

Greater part of the cases belonged to Pitha Kaalam which is commonly the period of degeneration (OP-90% & IP-85%).

4. Distribution according to Paruva Kaalam:

High incidence of the disease was in Elavenil Kaalam with 45% in OP and Muthuvenil Kaalam with 35% in IP.

5. Incidence withThinai:

Most of the cases reported were from Marutham (OP-85% & IP-100%).

6. Incidence with reference to Constitution of the Body:

Pitha Vatha Thegi were much affected (OP-55% & IP-60%)

7. Incidence with reference to Gunam:

All cases had Rajo Gunam.

8. Incidence with reference to Religion:

The highest incidence was found to be among Hindus (OP-95% & IP- 90%)

9. Incidence with reference to Socio-Economic Status:

In this clinical study most of the patients were from low income class with 70% in IP and also from middle income class with 50% inOP.

10. Incidence with reference to Food Habit:

Most of the patients belong to non-vegeterian (OP-90% & IP-70%).

11. Incidence with reference to Family History:

Most of the patients don t have family history related to thisdisease(OP-85% & IP-85%).

12. Incidence with reference to Occupation:

Most of the patients strained themselves as heavy workers, lifting heavy weight, travelling a long distance and sitting for a long period of time. These may be the reasons to develop Thandaga Vatham.

13. Incidence with reference to Aetiological Factors:

Age, obesity and occupation were the main precipitating factors in majorityof cases.

14. Incidence with reference to Mode of Onset:

Most of the patients were observed in chronic state (OP-90% & IP-95%).

15. Incidence with Duration of Illness:

Majority of the cases were observed above 12 months and 1-6 months of duration (OP-35% & IP-50%).

16. Incidence with Clinical Manifestation:

100% of both OP & IP had low back pain and exacerbation of pain on movements. Radiating pain to lower limbs, restricted movements, stiffness, tenderness and numbness were present in variable number among the patients under study.

17. Incidence with reference to Kanmenthiriyam:

- Kaal was affected among 85% of OP & 100% of IP.
- Eruvai was affected among 35% OP & 25% of IP.

18. Incidence with reference to Gnanendrium:

- Mei (Local heat) was affected among 25% of OP and 30% of IP.

19. Incidence with reference to Kosam:

- ❖ Vignanamaya Kosam (Restriction of movements, low back pain) was affected in allcases.
- ❖ Annamaya Kosam (Anorexia)was affected among 30% of OP & 25% of IP.

20. Condition of Mukkutram:

a) Disturbance in Vatham:

- ❖ Koorman was affected among 10% of OP.
- ❖ Kirukaran (Secretion of saliva) was affected among 30% of OP and 25% of IP.
- ❖ Viyaanan (Movements, nervous functions and sensation), Samaanan (Regulates the digestion and controls all the other vayus) and Devathathan (Laziness) were affected in allcases.
- ❖ Abaanan (Constipation) was affected among 35% of OP and 25% ofIP.

b) Disturbance inPitham:

- ❖ Sathagam (Wilful activities) was affected in all cases.
- ❖ Analagam (Digestion) was affected among 30% of OP and 25% ofIP.
- ❖ Ranjagam (Nutrition to blood) was affected among 25% of OP and 10% of IP.

c) Disturbance inKapam:

- ❖ Santhigam (Integrity of joints) was affected in allcases.
- ❖ Klethagam (Lubrication of food) was affected among 30% of OP and 25% of IP.

21. Incidence with reference to Udal Thathukkal:

Saaram, Kozhuppu, Enbu were affected in all cases. Senner wasaffected among 25% of OP and 10% of IP. Disturbance in Saaram produce symptoms like lethargy and mental depression. Disturbance in senner was associated with Anemia. Disturbance in Kozhuppu, Enbu produce restricted movements, reduced intervertebral disc space, extra osteophytic changes and degenerative spondylotic changes in LumbarVertebrae.

22. Incidence with reference to Envagai Thervugal:

- ❖ In this study all cases had thontha naadi with high incidence of Pitha Vatham (OP-55% &IP-60%).
- ❖ Sparisam was affected among 25% of OP and 30% of IP.
- ❖ Malam was affected among 35% of OP & 25% of IP.

23. Incidence with reference to NeerKuri:

- Niram was affected among 30% of OP & 35% of IP.

24. Incidence with reference to Neikuri:

Majority of cases showed the neikuri as spreading like snake, when the oil is dropped into the urine indicating the predominance of Vatha neer (OP-70% & IP-65%).

25. Incidence with reference to Radiological Studies:

From the X-Ray of Lumbar spine (AP & Lateral view) all the cases had degenerative spondylotic changes. Reduced lordosis was found among 10% of IP. Reduced intervertebral disc space found among 10% of OP and 15% of IP. After treatment no changes is noted in X-Rays.

26 (a) Assessment of Outcome-Cardinal Signs

After treatment, the clinical trial drug showed significant effect in improving the grades of cardinal signs of Out patients - low back pain, radiating pain, restricted movement, stiffness, tenderness and numbness at the high level of significance at P<0.001, 0.001, 0.01, 0.05, 0.001, 0.005respectively.

After treatment, the clinical trial drug showed significant improvements in the grades of cardinal signs of In patients - low back pain, radiating pain, restricted movements, stiffness, tenderness and numbness at the high level of significance at P<0.001, 0.001, 0.0001, 0.01, 0.05, 0.05respectively.

26 (b) Range of Motion

After treatment, range of movements score showed significant improvement among Out patient at the high level of significance at P<0.001.

After treatment, range of movements score showed significant improvements among In patients at the high level of significance at P<0.001.

26 (c) Back Pain Functional Scale Score

After treatment 65% of OP and 70% of IP had good improvement. 15% of OP had moderate improvement. 10% of OP and 20% of IP had mild improvement.

Bio Statistical analysis

The BPFSS, on after treatment show a strong evidence, that the clinical trial drug among the Out patients showed a positive relationship and is considered to be extremely statistically significant at P value less than 0.001 (t=18.1542 & 95% of confidenceinterval).

The BPFSS, on after treatment show a strong evidence, that the clinical trial drug among the In patients showed a positive relationship and is considered to be extremely statistically significant at P value less than 0.001(t=10.9651 & 95% of confidenceinterval).

27. Incidence with reference toresults:

- 65% of OP and 70% of IP had good response.
- 25% of OP and 20% of IP had moderateresponse.
- 10% of both OP & IP had poorresponse.
- It was found that the end of result showed good clinical improvement in grade with reduction of low back pain, numbness, and stiffness, radiating pain, tenderness and restrictedmovements.
- Improvement in range of motion score and low back pain functional scale score is alsofound.

The statistical analysis of the observational parameters clearly indicates that Munnai Ilai Kudineer is highly siginificant in the treatment of Thandaga Vatham.

Munnai Leaves has the taste of Thuvarppu, kaippu which regulates the vitiation of Pitham, lightens the body and maintains good health. Further it attains the kaarpu pirivu (section) which has the function of relieving indigestion, flatulence and constipation.

Leaves contains Verbascoside, Premcoryoside, Premnine, Premnazole, Premnenol, Premnaspirodine, Iridoidglycoside, benzoic acid which has anti-inflammatory, analgesic, anti oxidant activities. They are hepatoprotective and has anti microbial, anti bacterial and anti plasmodial potency (Jadhav Santosh et al.,).

Further, biochemical analysis of **MUNNAI ILAI KUDINEER** showed the presence of Sulphate, Chloride (Maintaining acid base balance), Ferrous iron (Increases haemoglobin level), Unsaturated compound (Building blocks of proteins) and aminoacids.

On pharmacological analysis, the anti-inflammatory and analgesic activity is due to β-sitosterol which blocks formation of prostacyclin, they inhibit cyclo oxygenase (COX) the enzyme that makes Prostaglandins (PGs) the flavonoids also inhibits the pain perception by inhibiting prostaglandins. Peripheral pain receptors to send a signal from the central nervoussystem.

Acute oral toxicity of Munnai leaves ethanolic extract given to wistar rats at the dose of 100mg/kg bw did not produce toxicities.

Infusion is the process of extracting chemical compounds or flavors from plant material in a solvent such as water, by allowing the material to remain suspended in the solvent overtime. Further, the herbs are immediately available for assimilation into the blood stream, glands and organs. Even patients with poor digestion and assimilation gets significant bio availability. So the constituents within the herb promote healing as well as maintenance of health (Anna Szymczycha-Madeja et al.,). So its best to uptake Munnai leaves asinfusion.

Thus Munnai Ilai Kudineer by virtue of its actions mentioned previously help in reducing the cardinal signs of lumbar spondylosis and prevents further degeneration.

CHAPTER-VII

SUMMARY

An open labeled Non randomized clinical study on **"THANDAGA VATHAM"** with reference to its aetiology, pathogenesis, clinical features, diagnosis, investigations and treatment were conducted at Department of Pothu Maruthuvam, Government Siddha Medical College Hospital,Palayamkottai.This clinical study of Thandaga Vatham is done on the basis of reference in**Yugi Vaidhya Chinthamani-800,** which is correlated in modern medicine is Lumbar Spondylosis.

The trial drug chosen for the clinical study is **MUNNAI ILAI KUDINEER** Dosage 30ml twice daily after food for 30 days (Ref.: Gunapadam Mooligai Vagupu, Page No.779-780).

A number of literatures were collected regarding Siddha as well as modern system of medicine.For this study, out of 40 patients, 20 patients were diagnosed clinically and admitted in the In patients ward and treated with trial medicines. Another 20 as Out patients

The patient treated with the trial drug MUNNAI ILAI KUDINEER twice a day daily for 30 days.Siddha diagnosis is achieved with the help of Envagai Thervugal and Ezhu Udal Thathukkal.

Since, Thandaga Vatham is a chronic disease, it requires treatement for minimum thirty days to minimize severe pain, tenderness, swelling and stiffness further the patient is advised to follow up the treatment in Out patients department.From this study the following datas are clear. The disease has increased incidence among individuals with more physical activity, higher BMI scores. Despite marked variability within the population, men appear to have more significant degenerative changes than women. Maximum incidence was in Kaba Kaalam. Clinically marked reduction in the symptoms along with increase sense of well being, **improvement in the grade of cardinal signs and range of motion,** decrease in the **"BACK PAIN FUNCTIONAL ASSESSMENT SCORE"** was noted.

The patients were observed for a period of 3 months during and after the course of treatment. No signs of complications were reported. Clinically no toxic effects were noticed during the treatment period. The Pharmacological evaluation and biochemical analysis of **MUNNAI ILAI KUDINEER** were also carried out results of this clinical trial study statistically and significantly proved.

CHAPTER-VIII

CONCLUSION

Thandaga Vatham is mentioned in Yugi Vaidhya Chinthamani-800 which is correlated with the clinical signs and symptoms are smillerly Lumbar spondylosis. The people suffering from lumbar spondylosis were increasing day by day even at the age of 30-60. The disease Thandaga Vatham cases treated with Munnai Ilai Kudineer and well analysed under Siddha and modern clinical parameters. In clinically MUNNAI ILAI KUDINEER highly response in 67.5%, showed good response; 22.5% showed moderate response; and poor response was 10%.By clinicaly pain score and flexibility were improved in case of lumbar spondylosis.

The review literature in siddha and modern journals are confirmed the action was MUNNAI ILAI KUDINEER was highly therapeutic effect of pain management in thandaga Vatham. In adition that invivo and invitro also detramined in MUNNAI ILAI KUDINEER is proved in analgesic (Table 5.1) , anti inflammatory action (Table 5.2), and anti microbial action (Table 5.5).

The presence of sulphate, chloride, ferrous iron, unsaturated compound and amino acid were found in Bio chemical analysis.

No toxic effect were noticed during the treatment period. It was confumed by In*vitro – In vivo* and acute and sub acute toxicity studies.

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ANNEXURE-I

PREPARATION AND PROPERTIES OF TRIAL MEDICINE

முன்னை இலை குடிநீர்

(Reference: Gunapadam Mooligai Part-I Page No.779-780)

Tamil Name	**Botanical Name (Family)**	**Phytochemicals**	**Action**	**Therapeutic uses**	**Amount**
Leaves of **Munnai**	*Prema corymbosa* (VERBENACEAE)	Verbascoside, Iridoid glycoside Premcoryoside Premnine, Premnazole Premnenol Premna spirodiene .	Anti-inflammatory, Anti-oxidant, Anti-analgesic	Vatha disease	7.5gms

PURIFICATIONS OF DRUG:

Fresh leaves of Munnai is cleaned to remove dust and impurities. Then, is made dry in shade.

METHOD OF PREPARATION:

Fresh leaves of Munnai is cleaned and made drying in shade. Then it is made into coarse powder. Now add 7.5gms of powder is boiled and make kashayam to dispensing for patients.

DOSE : 30 ML Twise\day

முன்னை இலை:

சுவை : துவர்ப்பு, கைப்பு

தன்மை : வெப்பம்

பிரிவு : கார்ப்பு

பொதுக்குணம்:

முன்னைவேர் வாதம் முதிரேப்பந்தான் போக்கும்
அன்னமிறக்கும் அதனிலைதான் - சொன்ன
இயல்வாதம் எண்பதையும் இல்லையெனச்செய்யும்
கயல்பொருவுங்கண்ணாய் கருது

முன்னை இலையை முறைப்படி குடிநீரிட்டு இருவேளை கொடுத்து வர வாதம் எண்பதும் தீரும்

முன்னை - Premna corymbosa

PREPARATION OF MUNNAI ILAI KUDINEER

DRIED LEAVES

MUNNAI ILAI KUDINEER

GOVT.SIDDHA MEDICAL COLLEGE
PALAYAMKOTTAI

SCREENING COMMITTEE

Name of the candidate : Dr.N.Prakash

Registration No: .....

DEPARTMENT OF POTHU MARUTHUVAM

This is to certify that the dissertation topic A Prospective open labeled Non Randomized phase-II clinical trial on herbal drug **"MUNNAI ILAI KUDINEER"** for the treatment of **THANDAGA VATHAM** (Lumbar Spondylosis) has been approved by the screening committee.

Branch	Department	Name	Signature
I	**Pothu Maruthuvam**	**Prof.Dr.A.Manoharan MD(S)**	
II	**Gunapadam**	**Dr.A.Kingsly MD(S) (Associate Professor)**	
III	**Sirappu Maruthuvam**	**Prof.Dr.A.S.Poongodi Kanthimathi MD(S)**	
IV	**Kuzhanthai Maruthuvam**	**Prof.Dr.D.K.Soundararajan MD(S)**	
V	**Noi Nadal**	**Prof.Dr.S.Victoria MD(S)**	for
VI	**Nanju nool Maruthuvam**	**Prof.Dr.M.Thiruthani MD(S)** For	

Place : Palayamkottai

Date : 26.05.2017

PRINCIPAL
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R.No.GSMC/5676/P&D/Res/IEC/2014 **Date: 29.05.2017**

CERTIFICATE OF APPROVAL

Address of Ethical Committee	Government Siddha Medical College, Palayamkottai, Tirunelveli(627002) district.
Principal Investigator	**Dr. N. Prakash**,MD(s) First year, Department of PothuMaruthuvam, Reg. No: Not yet registered.
Supervisor	**Prof.Dr.A.Manoharan, M.D$_{(s)}$.,** Head of the Department, Department of PothuMaruthuvam, Government Siddha Medical College and Hospital, Palayamkottai - 627002, Tirunelveli District. drmanoharan25@gmail.com
Guide	**Dr. T. Komalavalli, MD$_{(S)}$,Ph.D**,Asso.Professor, Department of PothuMaruthuvam Government Siddha Medical College and Hospital, Palayamkottai - 627002, Tirunelveli District
Dissertation Topic	A prospective open labelled non randomized phase II clinical trial to assess the therapeutic efficacy of the Siddha formulation **Munnai ilai kudineer** for the treatment of **THANDAGAVATHAM**(Lumbar Spondylosis)
Documents Filed	(1)Protocol (2)Data Collection Forms (3)Patient Information Sheet (4)Consent Form (5)SAE (Pharmacovigilance)
Clinical/Non Clinical Trial Protocol (Others-Specify)	Clinical Trial Protocol-yes
Informed Consent Document	Yes
Any other Document	Case Sheet/Investigation Documents
Date of IEC Approval & its Number	29.05.2017, GSMC-IV-IEC/2017/Br-I-07/29.05.2017

We approve the trial to be conducted in its presented form.

The Institutional Ethical Committee expects to be informed about the process report to be submitted to the IEC at least annually of the study, any SAE occurring in the course of the study, any changes in the protocol and submission of final report.

Chairman

(Prof. Dr.Murugesan M.D(s).,)

Member Secretary

29/5/17

(Prof.Dr.R.NeelavathyMD(s)Ph.D.,)

K.M. COLLEGE OF PHARMACY – MADURAI

IAEC – CERTIFICATE

This is to certificate that the project title **A PROSPECTIVE OPEN LABELED NON-RANDOMIZED CLINICAL TRIAL OF "MUNNAIILAI KUDINEER" FOR THANDAGA VATHAM (LUMBAR SPONDYLOSIS)** has been approved by the IAEC/ N.PRAKASH /TNMGRMU/MD(S)/321611007/KMCP/20/2018.

Dr. N. CHIDAMBARANATHAN

Name of the Chairman / Member Secretary IAEC:

Dr P. THIRUPATHY KUMARESAN

Name of the CPCSEA Nominee

Signature with Date 11/3/18

I. A. E. C. Chairman
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K. M. COLLEGE OF PHARMACY
MADURAI-625 107.

11/3/18

CPCSEA NOMINEE
INSTITUTIONAL ANIMAL ETHICS COMMITTEE
K.M. COLLEGE OF PHARMACY
MADURAI-625 107

Chairman / Member Secretary of IAEC

CPCSEA Nominee

(Kindly make sure that minutes of the meeting duly signed by all the participants are maintained by office).

GOVERNMENT SIDDHA MEDICAL COLLEGE
PALAYAMKOTTAI

Certificate of Botanical Authenticity

Certified the following plant drug used in Siddha formulation (Internal) **"MUNNAI ILAI KUDINEER"** for **"THANDAGA VATHAM"** (Lumbar Spondylosis) taken up for Post-Graduation Dissertation Studies by Dr.N. PRAKASH, PG Scholar MD siddha, Department of Pothu Maruthuvam, is correctly identified and authenticated through Visual inspection / Organoleptic Characters / Experience, Education & Training Morphology Microscopically and Taxonomical methods.

Table 1: Ingredients of Munnai Ilai Kudineer

S.N	Drug	Botanical Name	Family	Parts Used
01	Munnai Ilai	*Premna corymbosa*	Verbenaceae	Leaves

Station: Palayamkottai

Date : 13.2.18.

13/2/18

Authorized Signature

Dr. S. SUTHA, M.Sc.,M.Ed.,Ph.D.,
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The Tamil Nadu Dr. M.G.R. Medical University

69, Anna Salai, Guindy, Chennai - 600 032.

This certificate is awarded to Dr/~~Mr/Mrs~~.....N. PRAKASH.....

for participating as ~~Resource Person~~ / Delegate in the XXIII Workshop on

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The Tamil Nadu Dr. M.G.R. Medical University from 6th to 10th March 2017.

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Dr. N. Prakash

has participated in Continuing Medical Education on "**AYUSH External Therapies-II**" held at GSMCH, Palayamkottai on Dec, 4 2018

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Principal

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This is to certify that Dr...N...PRAKASH...B.S.M.S....M.D(S).....has participated in CME Programme on the topic **"PREPARATORY METHODS OF AYUSH MEDICINES TOWARDS GMP STANDARD WITH STANDARD OPERATING PROCEDURE FOR AYUSH PRACTITIONERS"** held by IMPCOPS jointly with RASHTRIYA AYURVEDA VIDYAPEETH from 18th-23rd, March 2019 at IMPCOPS, No.34-37, Kalki Krishnamurthy Salai, Thiruvanmiyur, Chennai - 600 041.

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IJMPR 2019, 3(3), XX-XX

International Journal of Modern Pharmaceutical Research
www.ijmpronline.com

ISSN: 2319-5878
IJMPR
Research Article
SJIF Impact Factor: 3.498

THE EVALUATION OF ANTI-INFLAMMATORY ACTIVITY OF SIDDHA FORMULATION MUNNAIILAI KUDINEER *(PREMNA CORYMBOSA, BURM.F.)* CARRAGEENAN INDUCED ALBINO WISTAR RATS

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Received on: 20/04/2019
Revised on: 10/05/2019
Accepted on: 30//05/2019

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ABSRACT

The Munnaiilai kudineer (MIK) is*(Premna corymbosa, Burm.f.)*used in Siddha system for the management of arthritis, gastric ulcer, hypertension, giddiness and hemorrhoids. The classical siddha text Gunapadam mooligai part 1 **(Dr.K.S.Murugesa Muthaliyar).** The aim of study is to evaluate the anti-inflammatory activity of Munnaiilai kudineer. Test compounds are assessed for acute anti-inflammatory activity by examining their ability to reduced incarrageenan-induced hind paw edema and pleurisy induced rat models.The end of result showed Munnaillai Kudineer behaves as an inhibitor of leukocyte migration and the formation of pleural exudates,after oral administration of MIK had significantly reduced in swelling in Wister albino rat.

KEYWORDS: Anti inflammatory, MunnaiIlai Kudineer, Wisterrats, Siddha formulation.

INTRODUCTION

Siddha medicine is a traditional system, it was originated from South Indian based system of medicine. The Siddha system is an ancient medicinal practices and spiritual disciplines as well as alchemy and mysticism. In Siddha system of medicine are commonly used in plant, mineral, and animal resources, which is acquired from the natural surroundings.

Munnaiillai (Premnacorymbosa) is a potent medicinal plant in the Siddha system. Traditionally the leaves are used in the treatment of vatha diseases, giddiness, loss of appetite and in pain management.

Inflammation is a complex biological response of vascular tissues against aggressive agents such as pathogens, irritants,or damaged cells. Acute inflammation is the initial responseand is characterized by the increased movement of plasmaand innate immune system cells, such as neutrophils and macrophages, from the blood into the injured tissues. The standard signs of inflammation are expressed by increased bloodflow, elevated cellular metabolism, vasodilatation, release ofsoluble mediators, extravasation of fluids and cellular influx (Ferrero-Milianet al.2007). Upon the presence of the inflammatory agent, cell membranes induce the activation of phospholipase A2 followed byrelease of arachidonic acid and inflammatory mediators such ascytokines, serotonin, histamine, prostaglandin and leukotrienes that increase vascular permeability, thus facilitating the migra-tionof leukocytes to the site of inflammation (Dassoler et al. 2004).

Inflammation induced by carrageenan is acute, nonimmune, well-researched,and highly reproducible. Cardinal signs of inflammation edema,hyperalgesia, and erythema developed after immediately following subcutaneous injection, resulting from action of proinflammatory agents,bradykinin, histamine, tachykinins, complement and reactive oxygen, and nitrogen species. The saponins displayed significant antinocicep-tive, anti-inflammatory and antipyretic activities possibly due totheir nonglycosidic moiety. The sapogenin is diverse activities havebeen reported such as antiallergic, antifungal, analgesic (Hostettmann et al. 2007, Milgate et al.1995, and Francis et al.2007). Moreover a variety of siddha formulation preparation have proved to be useful in animal models of inflammation [De La Lastra.C.A et al.2007, Liu Yet al.2012and Kang 2010].

Paw swelling or foot pad edema is a formidable method for assessing inflammatory responses to antigenic challenges and irritants. The test compounds are assessed for acute anti-inflammatory activity by examining their ability to reduce or prevent the development of carrageenan-induced paw swelling. In the present study attempts are made to validate the claims of Munnaillai Kudineer (MIK)regarding the anti-inflammatory activities.

International Journal of Reverse Pharmacology and Health Research (IJRPHR)

Research article

Toxicity study on Siddha formulation *Munnai Illai Kudineer* in Albino Rat

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Abstract

Munnailai kudineer(Premna corymbosa) is the Siddha formulation used in the treatment of vatha diseases and in pain magement. The aim of this present study is to evaluate the acute and sub acute toxicity of Munnaiilai kudineer(MK). Toxicity of Munnaiilai kudineer is carried out as per the guidelines Organization of Economic Co-operation and Development (OECD) -423 guidelines. The result of toxicity study shows that there was no significant change in animal behavior due to the absence of toxicity. The animals treated with MK showed normal growth pattern and body weight compared with control rats treated with normal saline. The overall results suggest that Munnaiilaikudineer(MK) are non-toxic to the haaematopoietic and leucopoietic system.

Keywords: Siddha medicine, Munnailai kudineer, Rat models.

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CODENJ : IJRPHR

To access this article online

Website : http://www.ijrphr.com/

DOI : 10.121/ijrphr/02.0203.315

Quick response code

Introduction

Siddha system is unique among the Indian system of medicine. It is believed to have been developed by the siddhar's the ancient supernatural spiritual saints of India.In Siddha system of medicine, the raw materials like plant, mineral, and animal resources are acquired from the natural surroundings. They have been used extensively for many centuries after thorough evaluation of the drug by traditional way. Siddha system emphasizes the dose regimen and pertinent vehicle for every medicine intake.

How to cite this article:

Prakash N, Manoharan.A, Komalavalli. T, Toxicity study on Siddha formulation *Munnai Illai Kudineer* in Albino Rat, International Journal of Reverse Pharmacology and Health Research, 2019, 2(1), 31-36

Received: January, 2019.

Accepted: March, 2019.

Clinical Trial Details (PDF Generation Date :- Tue, 02 Jul 2019 03:33:42 GMT)

CTRI Number	CTRI/2018/04/012990 [Registered on: 03/04/2018] - **Trial Registered Prospectively**
Last Modified On	03/04/2018
Post Graduate Thesis	Yes
Type of Trial	Interventional
Type of Study	Siddha
Study Design	Single Arm Trial
Public Title of Study	A clinical study to study the drug Munnai ilai kudineer on vatham
Scientific Title of Study	A prospective open labelled non randomized phase II clinical trial to assess the therapeutic efficacy of the Siddha formulation Munnai ilai kudineer for the treatment of Thandaga Vatham (Lumbar Spondylosis)

Secondary IDs if Any

Secondary ID	Identifier
NIL	NIL

Details of Principal Investigator or overall Trial Coordinator (multi-center study)

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URKUND

Urkund Analysis Result

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Sources included in the report:

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